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ANTERIOR POLIOMYELITIS, WITH SPECIAL REFERENCE TO PATIENTS PASSED THROUGH THE ROYAL ALEXANDRA HOSPITAL FOR CHILDREN IN RECENT EPIDEMICS.[1]

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I HAVE prepared this short paper at the invitation of the President and the Council, not as one who by intense study and research is able to make authoritative statements as to the epidemiology, pathology, symptomatology and treatment of the disease known as anterior poliomyelitis, but rather as one who has had close contact with the condition in both the 1928-1929 epidemic and the present one, totalling in all some 250 cases. I will therefore deal with the subject from the practical aspect, trusting that my remarks may in some little way assist the members to a fuller understanding of the symptoms and treatment of this infectious disease.

[1] Read at a meeting of the Old Sydney Hospitallers' Club on March 1, 1932.

Ætiology.

As you are well aware, poliomyelitis is an epidemic disease which has been known for many years, an epidemic having been described at Stockholm by Medin as early as 1887. It is normally a sporadic disease, but has assumed epidemic proportions in many countries—in England, in Europe, in America and in Australia. In this country it usually commences in the summer and exhausts itself by the coming of autumn. In 1928 it commenced in Sydney in October, reached its zenith in March of 1929, and faded out by June of the same year. The present epidemic commenced last year with a case in June, another in July, two in September, one in October, and in December we had our greatest number of admissions, being 45 for the

month. In January there were 36 admissions and during February, 21. Apparently the epidemic in the metropolis has almost exhausted itself, and we do not anticipate many more cases from now on. It is interesting to note, as is usual in these epidemics, that the country districts did not report many infections until late January and February, when the disease in Sydney was on the wane. You will have noticed that the epidemic this season occurred generally earlier in the summer than in the 1928-1929 epidemic. I have graphed the curve of the epidemics against the average maximum and minimum temperatures and the humidity over the periods of the epidemics, but have failed to find anything of value in so doing.

Sex Incidence.

The incidence of sex has revealed in the two epidemics a greater proportion of boys, in the 1929 epidemic the proportion being boys 57%, girls 43%, and in this epidemic boys 61%, girls 39%. This proportion of six boys to four girls is not the normal ratio of boys to girls born in New South Wales. I was supplied with figures by the Government Statistician on this subject and find that the mean average of boys to girls born in New South Wales during the past decade is almost equal, actually 105 boys to 100 girls.

Age Incidence.

Children of any age may be affected. This paper deals with patients admitted to the Royal Alexandra Hospital for Children, where the age limit is thirteen years, so that I am unable to give figures and make statements as to the frequency with which adolescents and adults are affected, but these we know to be much more rarely affected than children. In the earlier epidemic there were two cases in infants under a year old, while the years from one to four appear to have been particularly dangerous with respect to susceptibility. This season we have had eight patients under twelve months, while children one year old appear to have been especially infected. This seems to suggest a plausible explanation with respect to the age incidence in this present epidemic. Children who were alive during the 1928-1929 epidemic, but apparently did not become infected during the present epidemic, appear to have shown some immunity. Whereas, relatively, the greatest number of cases were amongst infants and children one year old, that is, those born since the last epidemic.

Symptoms.

During the present epidemic the well known symptoms of the onset were almost invariably noted. The onset is generally sudden, with fever up to 38·9° C. (102° F.) or thereabouts, nausea, drowsiness and irritability. Vomiting is a frequent symptom, diarrhœa is not so frequent, retention of urine, though very useful, is rather rare, and the older children complain of soreness and stiffness of the neck and back. The signs exhibited in the earlier stage are those of neck rigidity, drowsiness and irritability and spine sign, and in many cases muscular tenderness. Hyperæsthesia is a sign frequently mentioned with reference to the early recognition of anterior poliomyelitis, but we have not found it easy to elicit nor, generally speaking, of value. I have reviewed the symptoms and signs in about 120 cases with respect to the frequency of their occurrence and here present them.

Drowsiness or irritability occurred in nearly every case.

Fever occurred in 55% of cases.
Vomiting occurred in 50% of cases.
Diarrhœa occurred in 9%.
Retention of urine occurred in 5% of cases.
The spine sign was demonstrated in 50% of cases.
Neck rigidity was demonstrated in 56% of cases.

I wish to indicate that the figure 50% with reference to the spine sign does not emphasize its true frequency nor its real value, as over 25% of the 120 patients mentioned were admitted and examined some days after paralysis had been definitely established, when the spine sign (which is a sign particularly noted in the preparalytic stage) is rarely present. Taking this fact into account would show that the spine sign was demonstrated in about 70% of the early cases.

The Spine Sign.

At this juncture a description of the spine sign and another sign really dependent upon it, Amos's sign, might be of value. The spine sign is a very definite sign elicited, depending upon the age of the patient, in two ways. If an infant or a small child, it is best to raise the child from the bed with one arm under the neck and shoulders and the other behind the knees. On attempting to approximate the forehead to the knees, the child remains stiff, pain is inflicted and the child cries out. If the patient is a bigger child or an adult, seat him on an examination couch or the side of the bed and request him to kiss each knee in turn. This is impossible, the attempt causing obvious pain. The spine sign, which is a true sign that has to be elicited, must not be confounded with the rigidity of the back associated with loss of power of the lower limbs, frequently seen in patients, or with the sore and painful back not infrequently following lumbar puncture.

Amoss's Sign.

Amoss's sign or the "tripod" sign is frequently seen among the bigger children. When the patient is asked to sit up and is assisted into a sitting position, the hands are placed flat on the bed or the couch, behind the back, and the arms and back are kept stiff, thus forming a tripod. The appearance of a child demonstrating the tripod sign is quite unlike the hunched attitude of the normal child in the sitting posture, or the child in the early stages of other acute infectious diseases.

Want of Uniformity of Reflexes.

Another interesting feature noticed during this epidemic, and one that may even be a useful sign,

is a lack of uniformity in the reflexes. This is noticed, of course, in the older children, that is, those above the age of two or three years, and this want of uniformity is especially seen in the knee jerks and is very suggestive.

Course of Symptoms.

To return to the symptoms. After the initial febrile attack, which may last one, two, three or four days, if paralysis is to ensue, symptoms and signs of paralysis then manifest themselves. I say advisedly, if paralysis is to ensue, because, as you know, many cases of so-called anterior poliomyelitis do not progress to the stage of paralysis. The foregoing is the usual typical onset, but many cases are atypical in mode of onset, some commencing with convulsions, some with stupor and even unconsciousness. I recall one case which commenced with convulsions, to be followed by a two-day afebrile period of apparent normal health, and then paresis ensued. Many of the patients were admitted quite unconscious, simulating the appearance of a case of tuberculous meningitis in the last stages, cerebral hæmorrhage or fractured skull; all of them were undoubtedly suffering from anterior poliomyelitis. Two patients were actually found lying on the ground unconscious, their parents having had no knowledge whatever of any illness or malaise. The tendency in this type of case is towards complete recovery, and it is among these patients that we find most of our so-called abortive cases. A common mode of onset is termed the "dromedary" type, when the stage of initial fever is followed by a lapse of two, three or up to seven days of apparently normal health, to be followed in turn by another febrile attack of shorter duration, and then paralysis.

After paralysis has ensued the temperature generally falls, and in a few days the patient loses the signs of stiffness and soreness; but in some instances, the so-called "stepping" or "jump" type, the temperature may remain elevated or, if it has fallen, rise again, and further groups of muscles may become involved. I should like to instance the case of one boy in whom over fourteen days elapsed before full paralysis was established, the groups being affected in this order: neck muscles, muscles of the back, the intercostals, and then those of both arms and legs—a very unusual order.

It is interesting to note that every one of the 93 patients with anterior poliomyelitis who passed through the Royal Alexandra Hospital for Children and were reviewed in 1929, showed some paralysis, whereas this season about 30% of the patients admitted to the same hospital in whom the diagnosis of anterior poliomyelitis was established clinically or cytologically, had no paralysis or paresis on admission. Of these some developed paralysis after admission, though others at no time showed any signs of myelitis. This was due, doubtlessly, to the earlier recognition of the symptoms of preparalytic anterior poliomyelitis by the general practitioner and to the opportunity afforded the practitioner of this earlier diagnosis by propaganda and the education of parents.

Theories as to Mode of Infection.

At this stage some discussion as to the mode of infection might be of interest. The more or less accepted theory as to the means of entry of the virus into the human body is by the nose and throat, then by means of the blood stream to the nerve cells. Therefore, it is said that coryza, post-nasal adenitis, pharyngitis and tonsillitis are frequently seen in the early stages of infantile paralysis. With this statement one must agree, though pharyngitis, coryza and tonsillitis have not been a prominent feature of the preparalytic stage. Burrows, of America, in a paper recently published, has given the results of fifty autopsies on fatal cases of anterior poliomyelitis and has stated that in all cases definite evidence of lymphatic infection was discovered. He alleges inflamed Peyer's patches, enlarged inguinal, axillary and cervical lymphatic glands, and his conclusion is that infection is gastro-intestinal in origin, the mode of spread being from the intestine through the lymphatic system, finally into the lymphatic spaces between the membranes of the brain and cord. Thus neck rigidity, the spine sign, irritability and other signs of meningismus are caused by pressure by distended lymphatic spaces. Early paresis is caused by lymphatic congestion in the cord, and recoveries are due to the gradual removal of the pressure from these congested lymphatic spaces. This theory is very interesting and impressive, though investigation at the Royal Alexandra Hospital for Children has failed to reveal clinical evidence of glandular enlargement or *post mortem* evidence of lymphatic involvement.

Forms.

Anterior poliomyelitis may result in a myelitis or may entirely abort. The following forms have been recognized in the two epidemics under review: the abortive, the spinal, the bulbar and pontine, the cerebral, the cerebellar, the ascending and descending and the meningitic. The neuritic form described has not been noted.

The Abortive Form. It is necessary to recognize the abortive form of this disease in which there are the signs and symptoms of a general infection, fever, malaise, drowsiness and general weakness. The symptoms may be accompanied by sore throat, coryza and even diarrhœa. Meningeal irritation may or may not be present. Paralysis does not result, and it is of course well nigh impossible to recognize such a case unless during an epidemic. I consider that this form is really the initial stage of a disease inadequately named anterior poliomyelitis or infantile paralysis. The symptoms and signs suggested by the terms infantile paralysis and anterior poliomyelitis may be regarded almost as a late stage in the one disease or even as an unfortunate but frequent complication of the disease. Burrows, the aforementioned protagonist of the gastro-intestinal lymphatic mode of infection, goes so far as to suggest that anterior poliomyelitis is a misnomer, and that the disease might well be called "acute lymphatic hyperplasia".

The Spinal Form. The spinal form is by far the most common. It is characterized by a flaccid paralysis followed by wasting in the affected muscles. The muscles of the lower limbs are the most frequently affected, in the present epidemic constituting over 60% of the cases of paralysis. Next in order come the muscles of the upper limbs, and then the muscles of the face and neck, the back and the abdomen, the palate and pharynx and the intercostals. An interesting observation with respect to the muscles of respiration affected in anterior poliomyelitis, is that, whilst infantile paralysis generally involves the intercostal muscles, post-diphtheritic paralysis involves the diaphragm.

The Bulbar and Pontine Form. In the bulbar and pontine form the nerve cells affected are in the grey matter of the brain stem. The facial nucleus is quite frequently involved, there being ten such cases in the last epidemic. Affection of other nuclei in the brain stem account for paralysis of the sterno-mastoid muscles, the muscles of the palate, the larynx and the muscles of deglutition. In all but one of our twelve deaths respiratory paralysis was recognized.

The Cerebral Form. The cerebral form, next to the spinal form, is probably the most frequent. The symptoms of onset are as were described in the early part of the paper, and a variety of symptoms may result, depending upon the portion of the hemispheres affected. There are convulsions, choreic and athetoid movements, aphasia and slurring of speech, idiocy and lesser grades of mental impairment. These symptoms fortunately tend to complete disappearance.

The Cerebellar Form. There were only two instances of the cerebellar form noticed in the last epidemic, and these were manifested by tremulousness, general ataxia, and incoordination of movement.

The Ascending and Descending Form. The form described as ascending and descending, sometimes referred to as Landry's paralysis, does not occur very frequently. The paralysis moves generally in ascending order from the lower limbs upwards by a series of jumps or steps. One of these cases I have described to you. With respect to the term Landry's paralysis, it is now generally accepted that there is no such entity, and that the term has been applied to the "jump" type of anterior poliomyelitis.

The Meningitic Form. In the meningitic form one sees all the symptoms of meningeal irritation, even to the extent of marked head retraction. Paralysis rarely ensues and these patients have a tendency to a speedy and complete recovery.

The Neuritic Form. With respect to the neuritic form, I have never recognized a case and am quite unable to discuss its nature or the appearance of the patient so affected.

Diagnostic Biochemical Findings.

Although quite unable to discuss the detailed pathology or biochemistry of this disease, I wish to make mention of the cerebro-spinal fluid cell count and the blood leucocyte count.

The Cerebro-Spinal Fluid Cell Count. The cerebro-spinal fluid cell count is in my opinion, when taken in association with a knowledge of the presence of an epidemic and with the clinical picture, of extreme value. During the last epidemic, among some 90 patients in whom a cell count was done, there were only eight in whom the cells were below 4%, and these were generally represented by examinations of cerebro-spinal fluid done late in the disease, or particularly early in a very virulent form. Eight of the 90 patients examined had under 10 cells per cubic millimetre, but the general average cell count was about 80 cells per cubic millimetre. The count may be anything from eight or ten to 300 or 400 cells per cubic millimetre. In several cases it was even over 1,000 cells per cubic millimetre. One admits that the cells are increased in number in the cerebro-spinal fluid in such conditions as tuberculous meningitis, encephalitis, purulent meningitis and other diseases, but the low chloride content of a tuberculous meningitis, the presence of organisms in purulent meningitis, the clinical examination and a knowledge of the existence of an epidemic of infantile paralysis, when taken into consideration, prove the cell count to be a very valuable adjunct to diagnosis.

The Blood Leucocyte Count. The blood leucocyte count was not done in very many cases, but in those done it suggested that the leucocytosis is not very marked, being somewhere in the vicinity of 10,000 to 15,000 cells per cubic millimetre.

Diagnosis.

With respect to diagnosis I need add little to what I have already stated. The presence of an epidemic, the mode of onset, the presence of malaise, irritability, and the spine sign, and increased cells in the cerebro-spinal fluid, make diagnosis in most cases possible. The diagnosis of a sporadic case of anterior poliomyelitis is really a matter of great difficulty and doubt, unless paralysis is definitely established.

Prognosis.

The general prognosis in anterior poliomyelitis is really moderately good, as more than half of the patients completely recover after a lapse of time. In 1929, of 93 patients reviewed, 5 died, 17 were completely recovered on discharge from the hospital, and of the remaining 71 very few are now in any way crippled. Of the 120 patients reviewed during the present epidemic, 12 died, 42 were discharged from hospital totally cured, and 66 still had some paralysis or paresis remaining. Of these 66, about 40% to 50% are now only mildly paresed and should completely recover within the next six to twelve months, though the remaining number will probably have some residual weakness. With respect to the prognosis in the various forms, in those with spinal affection much depends upon the degree of nerve cell involvement of the cord. Among those with the bulbar and pontine form the facial paralyses

recover, while those with involvement of the vital centres almost invariably die, though three have recovered after intercostal muscle involvement. The prognosis in the cerebral, cerebellar and meningitic forms is very good, as most of these patients recover with very little or no stigma. The ascending or stepping form, generally accounts for considerable residual paralysis.

My more or less optimistic opinion as to the general prognosis may be contradicted by the perfectly true statement that most of the cripples in this State are crippled by infantile paralysis, but I think you will agree that many, if not most, of these cripples are of the type whose treatment, both the immediate early splinting and the later surgical follow-up treatment, has been rather neglected. I feel that with a clearer understanding of the condition by the physician and a wider education of the parents as to the necessity for maintenance of treatment and observation, infantile paralysis will not be directly responsible for as many cripples.

Treatment.

During the past epidemic, as you know, the treatment has consisted of the administration of convalescent anterior poliomyelitis serum. I do not feel justified in giving you at this stage an account of the serum administration at the hospital nor to express any opinions as to its use in the treatment of the disease. I will content myself with saying that it has been given free use for both preparalytic patients and those in the early stages of paralysis, and the final report of the Infantile Paralysis Committee on the subject should be both illuminating and interesting. Apparently serum must be given in the preparalytic stage only, though at no time will it prevent a fatal issue in a child dying of infantile paralysis. As to whether it will prevent a patient with infantile paralysis becoming one that is likely to die, I am not prepared to state. One is beset with great difficulties when considering the value or otherwise of the use of convalescent serum in infantile paralysis. The following are the reasons for this.

1. The difficulty of testing the potency of the serum used.
2. The lack at present of any means of standardization of dose.
3. The difficulty of judging the best mode of administration of the serum, whether intrathecally, intravenously or intramuscularly.
4. The difference in the virulence of the disease in different epidemics.
5. The different degrees of virulence in individual cases during a single epidemic.
6. The inability of comparing patients treated with convalescent serum with control cases of equal virulence and severity.

Another important and interesting aspect of serum treatment centres around the recent work of Burnet, of the Walter and Eliza Hall Institute, Melbourne, who has shown experimentally that Melbourne serum is impotent in monkeys affected with the Rockefeller virus. This suggests there are strains of virus, as there are strains of pneumococci or types of dysenteric bacilli, which, in treatment, will necessitate a serum prepared from their own particular strain.

Apart from serum treatment, the treatment of infantile paralysis in its early stages is rest of the affected muscles in the position of election. This is attained by intelligent splinting. The position of election is that in which the paralysed muscles or groups of muscles are completely supported and the antagonists assured of entire relaxation with no overstretching. Thus the use of Thomas's body splint for those patients fully paralysed, the modification of McIntyre's back splint, Isbister's more or less temporary splint, and even plaster of Paris, are of great value in this stage of treatment. The rest and splinting are maintained until one is certain that the active stage of infantile paralysis, as is indicated by further spread of tenderness of the muscles, has entirely disappeared. Then commences the next important stage, that of muscle reeducation. The value of the opinion of an experienced orthopædic surgeon in the early splinting treatment and the necessity of having the muscle reeducation conducted by a massage expert cannot be over estimated.

I should like to conclude with an expression of optimism with respect to serum treatment. The value of this treatment, as demonstrated experimentally on monkeys, is conclusive. The toxic virus of the cord of an infected animal, when injected as an emulsion, will cause death in a test monkey and will be counteracted by the administration of serum in a control monkey. I therefore feel that with a fuller understanding of dosage and mode of administration, convalescent serum will realize our hopes in the treatment of infantile paralysis.

THE PRINCIPLES OF AFTER-CARE IN INFANTILE PARALYSIS.

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THIS paper is addressed to those who have in their care patients convalescent from infantile paralysis. The first part of the treatment, namely, the giving of the convalescent serum, has been done wherever possible. Without doubt the results are much better than they would have been without it. But still we are left with a fair number of children and adults who have paralysed or partly paralysed muscles. There will be a natural tendency towards recovery in these cases. In many cases the recovery

will take place rapidly, but, speaking generally, a period of two years will elapse before the maximum recovery will have taken place. During this period proper treatment will assist natural recovery, but improper treatment or a policy of *laissez faire* will definitely thwart and defeat this tendency, and the patients will gradually become worse as time goes on.

Inadequate treatment results in a crop of deformities which will gravitate ultimately to the orthopædic surgeon for correction. This applies not only to the severe and moderate cases, but also to patients only slightly affected. Even one weak muscle will cause a deformity as surely as many weak muscles will. It is an orthopædic surgeon who now writes, but the duty of preventing deformity rather than correcting it falls upon the orthopædic surgeon. This duty he owes not only to the patient and the parents, but also to the community. It is worth while to dwell for a moment on the latter aspect. Of every hundred patients with infantile paralysis five, say, will die, and thirty will make early and complete recovery; sixty-five will be left more or less damaged. Without proper treatment thirty-five of these drift through stages of too early attempted use of unbalanced muscles into deformity and incapacity. Suppose twenty of these are unable to be supported by their parents, then these twenty will require a pound a week invalid pension for the rest of their lives. Suppose the average expectation of life in these to be forty years after they become invalided. Twenty pounds a week for forty years is forty thousand pounds. Within the last two decades the former hopeless outlook has given way to one of confidence, and it is not too much to say that properly directed and maintained effort would reduce these twenty State-maintained cripples to two.

There have been four hundred cases in the recent epidemic. Of those who are left convalescing from it no two will be alike. In detail the treatment will differ for each and every one, but the principles guiding the treatment will be the same in every case.

What are the principles involved in this adequate treatment? Briefly stated they are prolonged muscle rest in splints, interrupted only by daily muscle reeducation exercises. There is nothing here about massage or electricity, the ordering of which in the past was deemed to fulfil the obligation of the practitioner. Nothing in the world can lead a weak muscle back to strength except use of the muscle at the bidding of the will. No amount of massage, no electrical stimulation by themselves can add one iota of strength to a weakened muscle. As the accompaniments of voluntary effort they may occasionally be useful in a subsidiary way.

Muscle rest in proper splints is the prime necessity. It is necessary for two reasons: first, to shield the weak muscle from fatigue, and secondly, to prevent the stronger opponent muscle from acting to its detriment. A strong opponent muscle, if allowed to act at all, will produce bad effects on a weakened muscle. First, it stretches the weakened muscle and still further weakens it, and secondly, it deforms the limb by pulling it out of shape.

The first step to take is to make the complete test of each and every prime movement and an estimation of the relative strength of the muscles producing them. This is done according to the following scheme. In this scheme the muscle tested has been named in each instance after the directions for testing.

Muscle Testing.

Toe Movements: Patient lies supine on bed.

Flexion.	
Place examining finger under five toes and ask patient to press upon finger	Toe flexor
Extension.	
Place finger above four outer toes and ask patient to press finger up	*Extensor longus digitorum*
Repeat for big toe	*Extensor proprius hallucis*

Foot Movements:

Eversion.	
Hold finger one inch away from outer side of foot and ask patient to touch finger with foot	Peronei
Inversion.	
(*a*) With foot hanging down, hold a finger one inch away from inner side of foot and ask patient to touch finger with foot	*Tibialis posticus*
(*b*) With foot held up to a right angle, repeat as in (*a*) ..	*Tibialis anticus*
Plantar flexion.	
Ask patient to press foot downwards against resisting hand	Gastrocnemius and soleus

Lower Leg Movements:

Turn patient on side. Ask patient to bend knee	Hamstrings
Turn patient completely on face. Passively hold knee bent and ask patient to straighten, while an examining finger is held at ankle	*Quadriceps extensor*

Upper Leg Movements:

Patient lies supine.	
Examiner takes the weight of leg by lifting it by big toe. (*a*) Patient is asked to move leg outwards	*Gluteus medius*
(*b*) Patient is asked to move leg inwards	Adductors
Examiner lifts whole leg passively upward and patient is asked to press downwards on examiner's hand held behind ankle	*Gluteus maximus*
Ask patient to rotate whole leg internally	Internal rotators
Ask patient to rotate whole leg externally	External rotators
Patient turned to one side.	
Patient then asked to bend thigh forward	Ilio-psoas

Trunk Movements:	
Place examining hand on abdomen and ask patient to draw abdomen away from it. Watch for any inequality on either side	Abdominal muscles
Place two hands on either side of chest wall and ask patient to draw a deep breath. Judge the excursion of the chest wall	Intercostals
Prop patient up into half-sitting posture and ask patient to bend forward. (Watch if umbilicus is drawn to one side)	*Rectus abdominalis*
Lie patient face downward, with pillow under face. Then ask patient to clasp hands behind back and then raise head off pillow	*Erector spinalis*
Patient lies on back again.	
Ask patient to raise head off bed	Sterno-mastoid
Ask patient to whistle	Facial muscles
Ask patient to put out tongue	Lingual muscles
Shoulder Girdle Movements:	
Ask patient to shrug shoulders upwards	Trapezius
Ask patient to shrug shoulders backwards	Rhomboids
Ask patient to shrug shoulders forward	*Serratus magnus*
Upper Arm Movements:	
Lying supine.	
Place arm in nearly full abduction and ask patient to abduct completely	Deltoid, anterior, posterior
From abduction ask patient to adduct against resistance	*Pectoralis major* and *latissimus dorsi*
Bend elbow to right angle and ask patient first to rotate internally	Subscapularis
Then rotate externally	Infraspinatus and *teres minor*
Lower Arm Movements:	
Flex elbow	*Brachialis anticus*
Extend elbow	Triceps
Supinate forearm	Biceps and *supinator brevis*
Pronate arm	Pronators
Wrist Movements:	
Extend	Wrist extensors (radial and ulnar)
Flex	Wrist flexors (radial and ulnar)
Finger Movements:	
Extension at first interphalangeal joint	*Extensor communis digitorum*
Extension at second interphalangeal joint	Lumbricales
Flexion at first interphalangeal joint	*Flexor sublimus digitorum*
Flexion at second interphalangeal joint	*Flexor profundus digitorum*
Separating and approximating fingers	Interossei
Thumb Movements:	
Flexion	Flexors
Extension	Extensors
Roll thumb over towards little finger	*Opponens pollicis*

In recording the muscles use following formula: Figure 1 for muscles normal; 2, muscles capable of acting against gravity and resistance; 3, muscles capable of acting against gravity; 4, muscles capable of acting with gravity; 5, muscles capable of a "flicker" only; 6, muscles incapable of a flicker.

This record provides data both for the splinting required and the muscle reeducation exercises. The splints must be of such a kind that the "intelligent" muscles, finding it impossible for them to move, give up trying. Furthermore, they should not restrict the blood supply to the limb. For this reason an encircling plaster of Paris splint is not desirable. The splints of H. O. Thomas cover most of the ground and fulfil all the conditions. The basic splint is the hip splint, which should be used if any muscles of the hip, for example, glutei, or any muscles of the abdomen or lower part of the spine are affected. It secures fixation from the nipple line to the malleoli. If the foot be affected, it is necessary to add a right-angled foot splint which can be twisted to protect dorsi-flexors, plantar flexors, inverters or everters of the foot, as necessity dictates. If both lower limbs are affected, two such hip splints are joined together and made into a frame. If the upper part of the back or the neck is affected, a head-piece is added to the upper part of the frame. If the arms be affected, suitable supports to keep the upper arm abducted, the lower arm supinated or pronated, and the wrist fixed, according to their needs, can be attached to the frame. When the knee muscles are affected and the hip muscles unaffected, a bed knee splint suffices. When the arm alone is affected, an ambulatory abduction splint may be used. We can splint the neck separately by a Thomas's collar. When the facial or lingual muscles are affected, the help of the dentist must be procured to fashion appropriate splints. More and more it is becoming the practice of those who study the matter closely, to make use of the frame, even in patients who seem moderately or lightly affected.

It is better to keep these lightly affected patients fixed till they completely recover than to allow them to walk or to sit upright in the hope that use will strengthen the muscles. Too often we see it having the opposite effect.

The weight-bearing use of muscles, with or without braces, in the first year or so following infantile paralysis involving one or both legs, is risky and detrimental if practised to any considerable extent. By the avoidance of this and by the use of non-weight-bearing therapeutic muscular exercises conjoined with little or no walking in cases affecting the leg, it is possible, if the general principles already alluded to are followed out, to secure a class of results with which in the past we have been wholly unfamiliar. (Lovett.)

Instead of walking, swimming (especially in warm salt water), bicycling, and even crawling may be allowed.

The usual procedure in muscle reeducation is to release the child from the splint once a day six days a week, then, after a warm bath, in which extreme care is taken to keep the trunk and limbs in the same position they have occupied in the splints, the patient is brought to a table in a warm room. There the exercises are carried out; limbs which cannot be raised are asked to work on the horizontal, a powdered board being used to minimize friction. The exercises, which should be prescribed precisely and definitely by the surgeon beforehand,

are carried out according to the principles enunciated in Sir Colin MacKenzie's "Action of Muscles".

The necessary art and craft required to get them carried out, especially in very young children, can be attained only after a great deal of experience on the part of the masseuse. It is obvious that it will be necessary for the surgeon to change both the splints and the exercises from time to time to keep step with the changing progress of the muscles.

Sometimes muscles which are apparently totally paralysed can, by persistent effort on the part of the masseuse by coaxing the patient to send voluntary impulses through to them, be brought back to life and eventually to strength. Always partially paralysed muscles can be improved by these means.

For what length of time must these efforts continue? The answer is, up to full recovery, or for two years, or for longer than two years in the case of those muscles which are still showing some improvement. This seems a hard condition, but those who watch the results closely in a large number of cases, are convinced that to allow walking too early is to invite future lameness. The sooner the parents and patients are told of the possibility of two years' recumbency, the better for the peace of mind of all concerned. It is not too great a price to pay for fully recovered muscles and a limpless walk in the future.

THE PREVENTION OF DEFORMITIES IN THE TREATMENT OF POLIOMYELITIS.

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THE prevention of deformities is of paramount importance in the treatment of poliomyelitis. When there has been no treatment, disability results more often from deformity than from the paralysis itself. Moreover, while it is on the whole easy to prevent deformities, when they have once developed it is extremely difficult to correct them. The stretched muscles are at a disadvantage and cannot recover until the deformity is corrected, with the result that progress is prevented. Muscles which otherwise would have recovered, may, by prolonged stretching, have their power of recovery impaired or destroyed. Furthermore, the majority of the severely paralysed patients who do not wholly recover, can be made to walk with the aid of apparatus and crutches, provided they have been kept straight.

A deformity at one body level may affect other body levels. For instance, a fixed hip flexion may eventually produce flexion of the knee, equinus deformity, lordosis and scoliosis.

Deformities are avoided by anticipating their occurrence, from a knowledge of the causes which produce them, and by eliminating these causes if possible. Any threatened malposition should be over-corrected. Measures to avoid deformity should be undertaken as early as possible by immobilizing the patient in a hard bed. The limbs should be placed in the neutral positions; later, when the localization of the paralysis has become more definite, more exact measures to relax the affected muscles by over-correction may be begun.

Factors Leading to Deformities.

The factors which lead to deformities may be classified as follows:

(i) Disturbance of muscle balance.
(ii) Gravity.
(iii) Habit posture.
(iv) Static influences.

When, as a result of one or more of these factors, any malposition occurs or is maintained in a limb, the muscles of one group are thereby stretched and there is a tendency to contraction in the opposing group. Contraction gradually takes place in the muscles, fascia and ligaments, and the position becomes fixed.

Disturbance of Muscle Balance.

It is not the paralysis itself which produces malposition, but the loss of balance resulting from such paralysis. Total muscle paralysis around a joint does not of itself give rise to deformity, but to flail-limb. Even if the paralysis is moderately extensive, no deformity appears if the loss is equal in all directions and the balance is preserved. It is therefore important that we should have a complete knowledge of the muscle balance in every direction of each joint affected. From this we can forecast the likely malpositions. The slightest loss of balance must be carefully watched, and if any tendency to malposition is noted, such malposition should be over-corrected so that the weak muscles are favoured.

Deformities from slight imbalance may take months to appear. Danger lies in such an insidious onset; the weakness may be so slight as not to be detected early.

For example, at the Children's Hospital, a boy was thought to have recovered, but on presenting himself for review some months afterwards, a postural scoliosis was observed. The cause of this was a slight weakness of one sterno-cleido-mastoid muscle, which had resulted in a mild torticollis and a secondary scoliosis.

Such slight loss of balance may be detected in the following manner. Watch the attitudes taken by the other parts of the body when the patient is concentrating on a movement that needs effort. For instance, while the patient is lifting the leg from the bed, concentrating on hip flexion, note the attitude of the foot, the hands and the neck; you may thus be able to detect imbalance.

Gravity.

The action of gravity may increase a tendency to malposition, or even cause it. Gravity, even in a normal person, may determine a drop-foot, especially if aided by the weight of the bedclothes. If the dorsiflexors are weakened, the onset of an equinus

deformity is rapid. Gravity also rolls the lower limb outwards, placing a strain on the inward rotators of the hip. In the erect position the action of gravity in keeping the arms by the side will stretch a weakened deltoid. In the ambulatory patient weight-bearing will aggravate a slight malposition (for example, a valgus deformity).

Habit Posture.

Sitting.—If a child, paralysed in the leg, is allowed to sit up, the constant flexion of the hips and knees induces contractures of the flexors and stretching of the extensors. Even normal muscles may become seriously weakened under these conditions.

Lying.—When a child lies in a splint day after day, in a bed placed against a wall, and turns his head constantly in one direction, torticollis or scoliosis may be induced. To counteract this, the position of the bed should be altered frequently.

When the upper extremities are unaffected, constant use of the arms while the child is recumbent may develop the pectoral muscles. Tightness of the pectoral muscles and stretching of the posterior shoulder and scapular muscles result. This prevents the normal movements of bracing the shoulders backwards, upwards and outwards, and predisposes to round shoulders. Constant lifting of the child's head to watch the surroundings may lead to an exaggerated cervical curve or poked-out neck (ewe neck).

Trick Movements.—Trick movements, if unchecked, are likely to give rise to deformity. For instance, hitching of the pelvis by the lateral abdominal muscles on attempting abduction of the thigh, may lead to pelvic tilt. Extension of the wrist, when brought about by the flexors of the fingers, when the wrist extensors are weakened, shortens the flexors. Over-action of the trapezius, when the deltoid is lost, gives rise to hitching of the shoulder.

Static Influences.

Splint Deformities.—The splint in which the child is immobilized may be a potent cause of deformity. The two most common faults are failure to allow for growth, and improper bandaging. Children often grow rapidly, especially during the summer. If the foot-pieces of the splint are not lowered, knee and hip flexion, to accommodate the increased length of the limbs, will result. The legs may grow unevenly, causing pelvic tilt. The vertebral column also increases in length, and this, if not allowed for, results in the following:

1. The chest-piece of the splint becomes situated at too low a level. The child then may develop a high spinal curve.
2. The buttock and lumbar curves of the splint and child cease to correspond; some degree of lordosis is produced, which stretches the abdominal muscles.
3. The proper relation between the arm-pieces and the shoulder level is lost. This causes hitching of the shoulder and may produce *cubitus valgus* as well.

Improper Bandaging.—The bandages used to immobilize the child in the splint must be correctly and carefully applied, otherwise such deformities as *talipes equinus*, knock-knee or bow-leg are easily produced. The sling at the knee level should be correctly placed and, if necessary, a small soft pad should be added, just large enough to flex the knee slightly, to prevent hyperextension. Failure to do this is a frequent cause of trouble. Broken or bent splints will cause malposition. These must be looked for.

If a child appears with a deformity the beginning of which cannot be explained by the distribution of the paralysis, inspect the child's splint and watch the parent bandage the child in the splint. In the majority of cases the explanation of the malposition will become evident.

The Principles of Prevention.

In prevention the principles to be observed are: (i) Maintenance of the affected parts in the neutral anatomical position; (ii) over-correction of any threatened deformity.

By correction we not only prevent the deformity, but we favour the affected stretched muscles, thus aiding their recovery. Careful bandaging alone will in the majority of cases suffice to preserve the correct positions. If the deformity is not controlled by bandaging alone, either owing to the severity of the muscular imbalance or to the incapacity of the child's parent or attendant to follow instructions, a plaster mould splint should be made, holding the limb in the desired over-corrected position. This allows the limb to be removed daily for exercise and manipulation. Manipulation to prevent the onset of a deformity should be carried out at least twice a day. The parent should be given accurate instructions. There are certain principles for guidance.

1. Avoid injuring neighbouring joints. For example, when manipulating the foot, bend the knee to avoid straining the knee joint; such straining would produce, in this example, knock-knee or hyperextension.
2. Hold the limb close to the joint to avoid injuring the epiphysis.
3. Stretch the contracted structures by applying gentle force and using the three-point principle, which applies to the straightening of any curve.

In severe cases a complete plaster or a series of plasters is necessary. If a recurrence of a deformity is to be avoided, corrective precautions must be maintained until such time as the muscle balance returns, and when the patient begins to walk these must be continued by the use of apparatus, special boots and night splints.

The Likely Deformities.

I shall consider the most likely deformities that may appear in the various parts of the body, from any of the above reasons, and discuss their prevention. They must be treated along the general lines indicated above, but special points applicable to each will be mentioned.

The Foot.

Stubbing of the Toes.—When the child's foot outgrows the foot-piece, the toes are apt to become stubbed, especially if the bedclothes rest on the foot. Hammer-toes may result. The foot-piece of a splint or a plaster mould should always extend well beyond the length of the foot.

Hyperextension of the Great Toe.—Paralysis of the *tibialis anterior* muscle is a frequent occurrence; in such cases the *extensor hallucis longus*, which is the natural substitute for this muscle, is then used to dorsiflex the foot, and the big toe becomes hyperextended. During exercises for the *tibialis anterior* the toes should be held flexed to prevent hyperextension.

Talipes Varus.—Weakness of the peroneal muscles occasions *talipes varus*. This is prevented by full eversion of the foot. The inner border must be kept straight to correct the adduction. To avoid twisting of the foot, see that the heel and forefoot are equally corrected by the bandages.

Talipes Valgus.—*Talipes valgus* occurs when the invertors are paralysed. It is treated by full inversion of the foot.

Talipes Equinus.—Weakness of the anterior muscles and the action of gravity may determine an equinus deformity. The *tendo Achillis* contracts and a "tight heel" results. There may be some contraction of the long toe flexors as well.

In manipulating a foot affected with *talipes equinus*, avoid pushing the anterior end of the foot upwards and inducing a flat foot. Manipulate the foot in the varus position (except in the case of equino-varus).

Calcaneus.—When the *triceps suræ* is involved, care must be taken to provide plantar flexion early to avoid long-heel.

Combinations of the above-mentioned deformities are more usual: equino-varus, equino-valgus, calcaneo-valgus or calcaneo-varus. Should the *triceps suræ* alone be paralysed and the other plantar flexors escape, *pes cavus* may rapidly ensue, especially if the dorsiflexors are weak.

The feet should be carefully watched for the earliest signs of any of these deformities. Compare the two feet frequently. Alteration in the shape of the heel is an early indication of a commencing malposition, especially tight-heel or long-heel, varus or valgus. Institute the appropriate measures at once.

The Knee.

Knock-Knee.—Knock-knee is one of the commonest conditions to be guarded against. It may be present from previous rickets, but the most frequent cause is improper bandaging. It may be prevented by careful bandaging with a bandage which rolls the knee in and carries it outwards. Knock-knee is also caused by loss of muscular balance in the knee-joint, and is then often associated with outward rotation of the tibia. This movement of the tibia on the femur is produced by the biceps and the outer head of the gastrocnemius. The inward rotators are the popliteus, the inner hamstrings, and the inner head of the gastrocnemius. When these are affected the deformity may occur. The *tensor fasciæ latæ* muscle also plays a part in the production of this deformity. Contraction of this muscle, by virtue of its action on the ileo-tibial band, may produce outward rotation and eversion of the tibia, especially if there is slight knee and hip flexion. The opposite deformity, namely, inward rotation of the tibia on the femur, may occur when the outer group is paralysed; but this is much more uncommon, as it is normally a limited movement. A frequent cause of inward rotation of the tibia on the femur is paralysis of the inward rotators of the hip. The tibia is held in place by the foot in the foot-piece, and the femur is rolled outwards by the outward rotators of the hip and by gravity. Gravity acting on a varus deformity may also produce inward rotation of the tibia.

When knock-knee with outward rotation is threatened, three bandages should be used: one to control the knock-knee, one to roll in the tibia, and one to roll out the femur. Usually, however, a plaster mould must be constructed.

Hyperextension.—Incorrect splinting and paralysis of the hamstring muscles favour the production of hyperextension; but a weak gastrocnemius may also lead to this deformity.

The Hip.

Hip Flexion.—It is of great importance to prevent fixed flexion of the hip, as, when developed, this deformity is difficult to cure, and it delays the recovery of the *gluteus maximus*, a most important muscle in walking. It results in lordosis, pelvic tilt and knee flexion, and even in dislocation. The most common ætiological factor is sitting for prolonged periods.

The *tensor fasciæ latæ* is a muscle which often escapes when the other muscles of the lower limb are involved; and sitting, especially with a slightly abducted thigh, favours shortening in this structure particularly. The ileo-tibial band in these cases stands out when any attempt is made to correct the flexion, and the contraction may, as previously explained, be associated with knock-knee and outward rotation of the tibia. In the mild cases gentle stretching of the flexors, with the pelvis fixed and the limb adducted, serves to control the onset. The severe cases need prolonged treatment by a series of plaster splints, which hold the pelvis fixed, combined with traction on the affected leg. This is a long and difficult procedure. At the Children's Hospital we attribute the development of hip flexion often to the fact that the child is secretly allowed to sit up at home.

Adduction and Outward Rotation.—Deformities in the positions of adduction and outward rotation must be guarded against, as their presence increases the disability when walking is commenced.

Pelvic Tilt.—The pelvis forms the foundation of the body posture. Care should be taken, by frequent inspection, that the anterior iliac spines are kept level and that the spaces between the ribs and the

pelvic crest are equal on the two sides. Pelvic tilt is sometimes obscure in its origin, but is most frequently due to disturbance in the muscular balance. Weakness of the right abdominal muscles, *quadratus lumborum* and *erector spinæ*, allows the right side of the pelvis to drop. But the muscles attaching the pelvis to the lower limb also have an action on the pelvis, as the lower limb is kept fixed in the splint. Weak abductors of the right hip and weak adductors of the left hip allow the pelvis to drop on the left side. Various combinations of paralysed muscles occur. I have seen cases in which the two examples given have been present together without pelvic tilting. Other factors producing this deformity are: (i) shortness of one leg; (ii) habitual turning of the child to one side, which develops the lateral abdominal muscles of one side; (iii) the complete paralysis of one *gluteus maximus* when the other is normal, which causes the pelvis to drop on the side of the paralysed muscle. In the last case padding must be applied to the splint in order to level the patient. In the majority of instances pelvic tilt may be controlled by fixing the pelvis firmly to the splint by means of a wide pelvic grip strap, or by attaching the lower end of the corset to the splint at the level of the pelvis.

The Hand.

To prevent deformity of the hand, an accurate estimation of the muscles involved must be made. These are relaxed by immobilizing the hand on a *papier mâché* splint, moulded when wet to fit the patient, and subsequently dried.

The deformities likely to arise are: (i) the monkey-like hand, with loss of the web, due to loss of the *opponens pollicis* and *abductor brevis* muscles; (ii) hyperextension at the metacarpo-phalangeal joints and flexion at the inter-phalangeal joints; (iii) adduction of the first finger, from weakness of the first dorsal interosseous muscle; (iv) dropping forward of the fourth and fifth knuckles.

Opposition is the most important movement of the thumb, and special effort should be made to aid its recovery. Constant tricking of opposition, mainly by the action of the *flexor longus pollicis* and the *adductor pollicis* muscles, leads to flexion deformity of the terminal phalanx. The thumb should therefore be splinted in full opposition, with the terminal phalanx extended and the web wide. This aids the *abductor brevis* also. It is the action of this latter muscle which largely produces the rotation of the first metacarpal bone, seen in the movement of opposition.

When the lumbrical muscles are involved care must be taken to keep the fingers straight at the interphalangeal joints and flexed at the knuckles.

The Wrist.

Ulnar deviation, radial deviation and drop-wrist are the most common malpositions at the wrist to be avoided.

The Elbow.

The elbow is not commonly the site of deformity. Occasionally I have noted cases in which the growth of the arm had not been allowed for by altering the arm-piece. A slight degree of *cubitus valgus* resulted.

Pronation Deformity.

Pronation deformity is disabling, and is often rapid in onset and difficult to control. It arises from weakness of the *supinator brevis* muscle and indirectly from weakness of the elbow flexors. Paralysis of the biceps alone does not cause pronation deformity, but when the brachialis is affected as well, trick-flexion of the elbow by the *pronator teres* and the long flexors of the wrist and fingers may result in this malposition. In this case the elbow should be well flexed and the forearm bandaged in full supination; but in these cases a plaster mould splint to include the hand is usually needed to preserve the correct position

The Shoulder and the Scapular Region.

The deltoid is the muscle most frequently involved in the shoulder region. It recovers with difficulty. Abduction of the arm to a right angle is necessary. This is more than a neutral position, but it is used in order to favour the deltoid as much as possible and to prevent the occurrence of an adductor contraction. In spite of this precaution adductor contraction may occur. If an attempt is made to abduct the arm when the deltoid is paralysed, there is an immediate upward rotation of the scapula by the *serratus anterior* muscle, which normally does not act until the arm reaches a right angle. When, as often occurs, the rhomboids are weak as well, there is excessive rotation of the scapula, so that its inferior angle becomes visible in the axilla and the vertebral border may even assume a horizontal position. This predisposes to adductor contracture, even though the arm be held at right angles in the splint. A special corset with a small pad to hold the scapula back, and a large pad for counter pressure on the chest wall opposite, will be needed. This gives the over-stretched rhomboids a chance to recover and prevents the contracture of the adductors. If the *serratus anterior* muscle is paralysed, a similar pad, differently placed, may be used to keep the scapula close to the chest wall.

When weakness of the deltoid, supraspinatus and infraspinatus muscles is extensive, subluxation of the head of the humerus is likely. In the erect posture the weight of the arm would bring this about. In the splint excessive abduction will favour subluxation owing to the pull exerted by the *latissimus dorsi* and the pectoral muscles. Moderate abduction with full external rotation will correct this tendency.

Tightness of the Pectoral Muscles.

The arm-pieces of the splint are apt to be bent forward during the transport of the patient about the house or to the hospital. This, if uncorrected, predisposes to tightness of the pectoral muscles.

The Trunk.

The prevention of scoliosis is an important task in the treatment. Space permits of only a brief description. Paralytic scoliosis often commences very insidiously, but once it has commenced, it generally progresses rapidly; it may continue in spite of the recovery of the muscles originally affected. When established, scoliosis is incurable; early recognition is therefore important. The back should be examined frequently in every case of poliomyelitis. The deformity follows most frequently upon unilateral paralysis of the abdominal muscles or greater weakening on one side than the other. Some degree of paralysis of the abdominal muscles is very common in poliomyelitis. Of two hundred cases of the 1931 epidemic at the Children's Hospital, in 54% the abdominal muscles were involved. Lovett reports 72% in a large series of cases in New York in 1916. Other causes are unilateral weakness of the back muscles or of the intercostal muscles or of one psoas or of one *quadratus lumborum*. It may also follow: (i) unilateral deltoid paralysis, especially if the patient walks early and wears an unsuitable deltoid splint; (ii) an uncorrected shortening of one leg. The preventive treatment of scoliosis must be begun at the earliest stage. The period of immobilization and exercises must be prolonged while there is still any muscular imbalance, however slight, which would result in curvature. Paralytic lordosis and kyphosis may occur. These also are preventible by early prophylactic treatment.

The Neck.

The prevention of deformity in the neck, when the neck muscles are involved, may be difficult. An accurate estimation of the degree of muscular involvement must be made. The flexors and the extensors of the head proper and the posterior and anterior muscles of the neck should be tested separately. The latter are divisible into two groups, the sterno-cleido-mastoids and the deep neck flexors.

A frequent condition encountered is weakness of the *recti capiti anteriores* and the *longus colli*, with normal sterno-cleido-mastoids. This results in a characteristic deformity. The cervical vertebral curve becomes exaggerated, the neck thereby becoming shorter, the throat more prominent and the cervical concavity more marked. Contraction of the dorsal cervical structures may fix this position rapidly.

The child should be provided with a head splint with side checks. Small soft pillows are placed under the upper dorsal region and the head, to decrease the cervical curve and flex the head. In one case it was necessary to apply a plaster mould splint to hold this position.

If lateral imbalance is detected, measures are taken to insure that the child does not turn the head to the strong side. This may be effected by placing the bed against the wall so that the child's interests lie towards the weak side.

It must be remembered that, especially in the early weeks after the onset of the paralysis, when many muscles are recovering rapidly, the situation may change quickly and necessitate alteration in the corrective measures. For example, a foot severely paralysed may need to be put up into a valgus position on account of a threatened varus deformity. When the patient is next seen it may be noted that the peroneal muscles have responded to the relaxation and have recovered to such an extent that the balance is in their favour; in order, then, to avoid a valgus deformity, the foot needs to be placed in a varus position.

A NOTE ON THE SIGNIFICANCE OF DOSAGE IN THE IRRADIATION OF INTER-RELATED TISSUES.

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Introduction.

IN the radiotherapeutic treatment of malignant tumours we are essentially concerned with the irradiation of a nest of malignant cells which are themselves situated in normal healthy structures.

The success of the technique depends upon the fact that the radiosensitivity of the malignant cells is, in general, greater than that of the surrounding healthy cells; the ideal technique is the one in which the difference in radiosensitivities[1] is employed to greatest advantage.

The object of this note is to show that in any such combination of tissues, there is always one value of the dosage for which the difference between the biological effects in the two tissues will be a maximum.

Analysis.

In problems relating to the irradiation of cells and tissues it is known[1-10] that the relation between the fractional cellular survival and the dose of radiation administered takes the form:

$$P_1 = e^{-\lambda_1 q} \sum_{r=0}^{n} \frac{(\lambda_1 q)^r}{\lfloor r}$$

where P_1 = the fractional cellular survival,

λ_1 = the probability of absorption of one quantum of energy in the sensitive zone or organ when unit quantity of radiation is administered,

q = the dose of radiation administered,

$n+1$ = the minimum number of absorbed quanta required to destroy (or otherwise modify) a cell.

If now we simultaneously irradiate two superimposed or inter-related tissues of different radiosensitivities, the survival equations in their most general forms may be written:

[1] This difference in sensitivities is technically known as the radio-sensitive interval.

$$P_1 = e^{-\lambda_1 q} \sum_{r=0}^{n} \frac{(\lambda_1 q)^r}{\lfloor r} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (1)$$

$$P_2 = e^{-\lambda_2 q} \sum_{r=0}^{m} \frac{(\lambda_2 q)^r}{\lfloor r} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots (2)$$

where $\lambda_1 > 0$
and $\lambda_2 > 0$

If y represents the difference between the biological effects in the two tissues, we have:

$$y = e^{-\lambda_1 q} \sum_{r=0}^{n} \frac{(\lambda_1 q)^r}{\lfloor r} - e^{-\lambda_2 q} \sum_{r=0}^{m} \frac{(\lambda_2 q)^r}{\lfloor r}$$

and $$\frac{dy}{dq} = y^1 = -\lambda_1 e^{-\lambda_1 q} \frac{(\lambda_1 q)^n}{\lfloor n} + \lambda_2 e^{-\lambda_2 q} \frac{(\lambda_2 q)^m}{\lfloor m}$$

Now when $q = 0$,
$y = 0$
and as $q \to \infty$
$y \to 0$;
therefore y^1 must vanish at least once between 0 and ∞ and $y^1 = 0$

when $e^{(\lambda_2 - \lambda_1)q} = \frac{\lfloor n}{\lfloor m} \frac{\lambda_2^{m+1}}{\lambda_1^{n+1}} q^{m-n}$

Several cases now arise for consideration.

Case I. $\lambda_1 > \lambda_2$, $m > n$.

In this case we see that $e^{(\lambda_2 - \lambda_1)q}$ decreases from 1 to 0 as q increases from 0 to ∞ and $\frac{\lfloor n}{\lfloor m} \frac{\lambda_2^{m+1}}{\lambda_2^{n+1}} q^{m-n}$ increases from 0 to ∞ as q increases from 0 to ∞.

Hence there exists a unique ξ such that
$$y^1(\xi) = 0,$$
and in this case y^1 has only one zero and
$$y = 0.$$

The graph of y as a function of q is shown in Figure I.

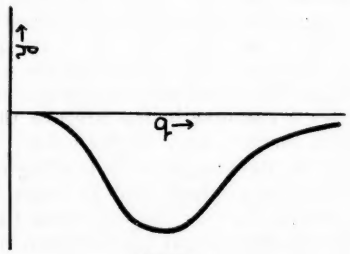

FIGURE I.

It is seen that as the dose of radiation increases from 0 to infinity the difference between the biological effect in the two tissues decreases from zero, attains a minimum value, and then increases again to zero. The value of q, producing the maximum difference in biological effects, can be easily calculated in terms of the characteristics of the irradiated tissues.

Case II. $\lambda_1 > \lambda_2$, $m = n$.

In this case it is clear that $\frac{\lfloor n}{\lfloor m} \cdot \frac{\lambda_2^{m+1}}{\lambda_2^{n+1}} < 1$ and there exists a unique solution as in Case I.

Case III. $\lambda_1 < \lambda_2$, $m > n$.

In this case $e^{(\lambda_2 - \lambda_1)q}$ increases from 1 to ∞ as q is increased from 0 to ∞, and it ultimately surpasses any power of q. We also know that y^1 must have at least one zero value, and hence the curves

$$y_1 = e^{(\lambda_1 - \lambda_2)q}$$

and $$y_2 = \frac{\lfloor n}{\lfloor m} \frac{\lambda_2^{m+1}}{\lambda_1^{n+1}} q^{m-n}$$

must cross an even number of times. Thus y^1 has an even number of zeros, and the graph of y must cross the axis of q at least once, and when $q = \infty$, y is clearly positive.

This case is represented diagrammatically in Figure II.

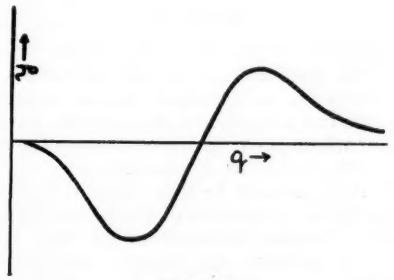

FIGURE II.

It is seen that as q increases from 0 to ∞, the difference between the biological effects decreases from zero, attains a minimum value, increases through zero to a maximum value, and then decreases again to zero. This result means that the survival curves given by equations (1) and (2) must cross, and as before, the values of q producing the maximum or minimum difference in biological effects can be easily calculated in terms of the characteristics of the irradiated tissue.

Case IV. $\lambda_1 < \lambda_2$, $m = n$.

From the previous work it is clear that in this case y^1 has only one zero value, and the relation between y and q can be represented diagrammatically as in Figure III.

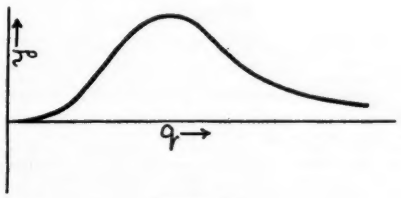

FIGURE III.

Case V. $\lambda_1 = \lambda_2$, $m > n$.

In this case we see that
$$y^1 = 0$$
when $\frac{\lfloor n}{\lfloor m} \frac{\lambda_2^{m+1}}{\lambda_1^{n+1}} q^{m-n} = 1$,

and a unique solution again clearly exists. The relation between y and q can be represented diagrammatically by Figure IV.

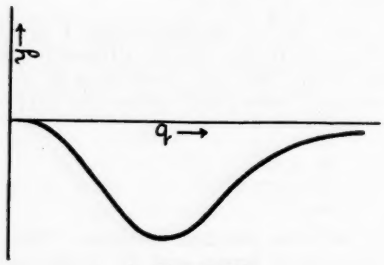

FIGURE IV.

Case VI. $\lambda_1 = \lambda_2$, $m = n$.
In this case it is clear that
$$y \equiv 0.$$

Discussion.

An inspection of the diagrams shows us that, for any possible combination of cells or tissues (except that combination of identical tissues considered in Case VI) we can always find at least one value of the dosage, for which the difference between the survivals will be a maximum or a minimum.

Before it is possible to determine this dosage, we must know the sensitivities of the tissues in question. Very little precise information of this nature is available at present for human tissues, but the determination of sensitivities is a fairly straightforward problem.

In view of the possible importance of this result in the radiotherapeutic treatment of malignant disease, it may be important to stress the fact that no hypotheses have been introduced into the above analysis. It is based entirely on experimental facts.

The result follows, in a general way, from simple considerations. The shape of the curve relating dosage and quantitative biological effects in any one tissue is always sigmoid. The difference between the quantitative biological effects of the radiation in inter-related tissues will, therefore, be graphically represented by the algebraic difference between two such sigmoid curves. This difference curve will always be of the same general shape as the curves already considered.

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METHOD OF DRAINAGE WITH IRRIGATION AFTER PROSTATECTOMY.[1]

By JOHN MORTON, M.B., Ch.M. (Sydney),
Honorary Consulting Surgeon, Royal Prince Alfred Hospital, Sydney.

THE main purpose of the paper is to describe a method of drainage after prostatectomy which I have used for some time past with satisfaction and which, I think, has several features to commend it. It is safe, easy of application, comfortable to the patient, permits of continuous bladder irrigation, and at the same time facilitates immediate closure of the suprapubic wound.

Before describing it, I should like to make a few general observations with regard to the operation. Apart from the question of renal efficiency, the chief dangers of prostatectomy are those which menace the success of any serious surgical procedure, namely, hæmorrhage, sepsis and general anæsthesia. The method of operation which best eliminates these dangers, is the one which will be most successful in its results.

General anæsthesia, especially if prolonged, is a real danger, and if the patient be at all of the "risky" type, there is nothing like spinal anæsthesia as far as my experience goes. If necessary, some 2% "Novocain" solution can be injected as well. In this connexion I should like to express my disapproval of adrenalin in the solution. I look upon it as quite unnecessary and often the cause of acute distress to the patient. Furthermore, I believe there is a greater liability to reactionary hæmorrhage after its use.

For rapid removal of the prostate there is great advantage in the bimanual method, with two fingers of one hand in the rectum.

The bimanual method, with two fingers in the bladder, is equally helpful in perineal prostatectomy, and the latter method can be very profitably resorted to when a small, tough, fibrous prostate is encountered in the course of a suprapubic operation. In both cases a catheter in the urethra throughout the operation forms a very useful guide.

Time spent in the complete controlling of hæmorrhage is not to be grudged, and at times a thermocautery is useful in this respect when ligature or oversewing with fine catgut has not been completely satisfactory.

It is very undesirable, however, to prolong the operation unduly, especially under general anæsthesia, and time may be saved by omitting any "toilet" of the prostatic cavity. I am of opinion that such is not essential to the complete success of the operation; no more so, I should say, than it is to close the cavity left after tonsillectomy.

It is highly desirable to get the bladder incision closed up as early as possible, but to do this immediately and depend on a catheter for drainage

[1] Read at the second annual reunion of the Residents' and Ex-Residents' Association, Royal Prince Alfred Hospital, Sydney, October, 1931.

does not appeal to me as the most satisfactory method. A catheter is liable to become blocked, and a temporary block in the catheter usually leads to leakage through the suprapubic wound. This frequently entails sepsis and sloughing of tissue, with delayed union and often a subsequent hernia.

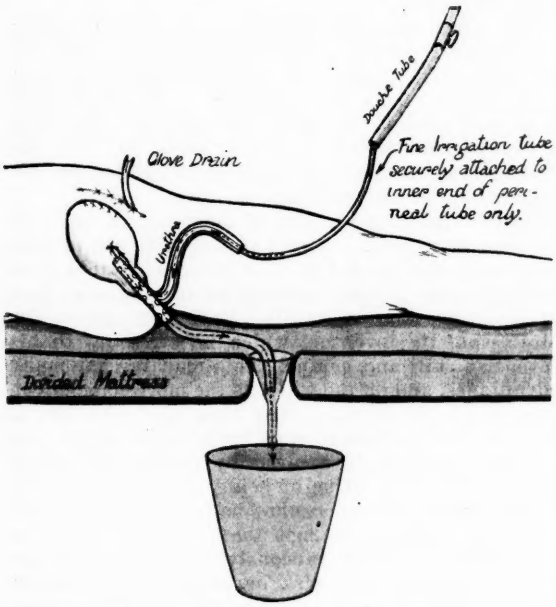

FIGURE I.

It may become necessary to remove the catheter in order to restore drainage. This is a very uncomfortable business for the patient, and it is not always an easy matter for the surgeon to reinsert a catheter without causing bleeding or damage of some sort. Failure of drainage may make all the difference between rapid and easy recovery or a retarded and painful one.

I feel satisfied that it adds greatly to the patient's comfort to have a temporary drain through the perineum. In this way the urethra, which is often very sensitive and intolerant of a large catheter, gets a complete rest, much more efficient drainage is afforded, and, moreover, continuous irrigation can be carried on through a fine, soft, flexible tube in the place of the catheter. It is a very simple matter to insert a perineal tube. With the patient held in the lithotomy position, make a small mid-line incision in the perineum behind the bulb. Shove a closed clamp through into the prostatic pouch, guided by two fingers of the left hand passed into the bladder from above. The track is dilated by opening the clamp, which is then used to grasp and pull down a perforated drainage tube through the suprapubic opening. This is fixed at the desired level by a silkworm gut suture to the skin of the perineum.

The fine irrigating tube is then drawn out through the urethra by attaching it to the end of the catheter which has been lying in the bladder.

The bladder end of the fine irrigating tube is closed securely by ligature and anchored to the bladder end of the perineal tube. Some tiny slits are made to allow the irrigating fluid, preferably saline, to flow into the bladder. This is important, because if the distal end of the fine tube be left open, blood may enter and block it. No reflux of blood can occur through the small slits, however, and the outflow of irrigating fluid under pressure is not hindered.

The suprapubic wound is carefully closed in layers, a strip of rubber tissue drain being left in the prevesical area as a precautionary measure.

When the wash-out is quite clear and the patient's condition otherwise satisfactory, the perineal tube can be removed, and the irrigation tube attached is easily withdrawn at the same time.

The drainage can be carried away by means of a funnel through the mattress or by tubing over the bedside into a bucket on the floor (Figure I).

THE SCOPE AND UTILITY OF GAS-OXYGEN ANÆSTHESIA.[1]

By H. J. DALY, M.B., Ch.M. (Sydney),
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History.

NITROUS oxide has been used by dentists in Sydney for many years past, but as it is so little known to Sydney surgeons I feel I must say something of its early history.

Priestley was the first to collect and examine the properties of "nitrous air", as he called it, in 1772; but Humphry Davy, in 1800, was the first to discover its anæsthetic properties, when he inhaled some for the relief of his own toothache. We next hear of it in 1844, when a Dr. Colton, in the United States of America, gave a popular lecture on nitrous oxide. A member of the audience, on inhaling the gas, wounded himself, but was unaware of this until he saw he was bleeding. A dentist named Wells noticed this fact and persuaded Dr. Colton to administer gas to him on the following day. While Wells was under the influence of the gas another dentist named Rigg successfully and painlessly removed a molar tooth. Wells was so impressed and delighted that he introduced nitrous oxide into his own practice. He made his discovery known to the medical men at Boston and obtained permission to extract a tooth under nitrous oxide anæsthesia at the local hospital. Unfortunately, as frequently happens at these demonstrations, the operation was not a success, and he was hissed out of the room and declared a humbug. This failure so preyed on his mind that he became insane and ended his days in a lunatic asylum.

This early misfortune caused nitrous oxide to fall into disrepute. The reasons are obvious. First, the

[1] Read at the second annual reunion of the Residents' and Ex-Residents' Association, Royal Prince Alfred Hospital, Sydney, October, 1931.

nitrous oxide was not always pure, and, secondly, the most primitive apparatus had to be used. Dr. Evans, who introduced nitrous oxide into England, had to content himself with an ox-bladder as a gas container. From this the gas was led to the patient by a wooden tube. In order to exclude the diluting air, the patient's lips were tightly compressed round this tube and the nose was unceremoniously pinched. Little more than analgesia could be produced. The difficulties of successful administration may be imagined.

In more recent times, it was not until the latter years of the Great War that the value of nitrous oxide and oxygen came to be recognized. It was greatly in demand for the shocked and bloodless patients. Perhaps its greatest value was when repeated dressing of painful wounds was necessary.

Scope.

An opinion generally held is that nitrous oxide can only be used for operations of the shortest duration. This idea originated from the old-fashioned "single dose" method. In this the anæsthetic was pushed to the stage of deep cyanosis, and the dentist had about forty seconds of anæsthesia in which to do his work. Such a method is difficult and crude, and not without risk to the patient. With modern apparatus and technique, nitrous oxide and oxygen anæsthesia can be maintained for hours if necessary.

Some Properties of Nitrous Oxide.

Nitrous oxide is generally prepared by the action of heat on ammonium nitrate. It is a colourless gas, heavier than air, having a density of about 1·5. It has a faint earthy smell, not at all disagreeable. At a pressure of 30 atmospheres it condenses to a colourless liquid; as such it is kept in metal containers. These containers are cylindrical and may be obtained in various convenient sizes, ranging from a capacity of 90 to 9,000 litres (20 to 2,000 gallons) of gas. Roughly, 424 grammes (15 ounces) of liquid nitrous oxide yields 220 litres (50 gallons) of nitrous oxide gas. Each cylinder has its weight (empty) stamped upon it, so that by weighing a cylinder and subtracting the weight of the empty cylinder, one can easily find out how much gas remains in any given cylinder after an administration.

Nitrous oxide has now been brought to a high standard of purity, and manufacturers preparing it for anæsthetic purposes put it to severe tests before delivery. However, it is a good plan to allow a gallon or so to blow off the top of the cylinder before use, as a precaution against the possible effects of any dangerous gases it may contain, such as carbon monoxide. Small quantities of water are occasionally found in a cylinder. This may cause obstructions to the flow of gas through the valves of a machine and necessitate the use of a hot water bag on them. This may occur even though most manufacturers stamp on their cylinders "Dry Nitrous Oxide". Nitrous oxide is non-inflammable and can be used safely in the presence of a cautery or diathermy machine.

Apparatus.

Progress in gas-oxygen anæsthesia has been very largely influenced by the perfection and improvement of different mechanical apparatus for its administration.

The latest types of machines evolved come under two classes: (i) the continuous flow type, and (ii) the intermittent type. In the first the gases flow continuously from the cylinder to the individual bags, and then to the mask and patient. The rate of flow is controlled by the anæsthetist. In the second type the flow of gases is controlled by the respiration of the patient.

After trial of a number of machines of the continuous flow type, I found it was difficult to give a smooth nitrous oxide anæsthetic with this kind of apparatus. I then secured an intermittent type of machine, known as the McKesson, after Dr. McKesson, its inventor, which is a tribute to his inventive skill and genius. This machine has many advantages, and on the whole is the more economical. The supply of gases coming from the cylinders is held in the individual bags by a single lever, and positive pressure is secured by merely tightening up a screw. The value of being able to deliver the gases under pressure cannot be over-estimated, one great advantage being that oxygen can be administered instantaneously and the lungs inflated without any risk or possibility of injury. This is due to the fact that the oxygen escapes under the edges of the rubber face-piece when the lungs are fully inflated, and thus over-distension and injury are prevented. An ether container attached allows one to add any desired quantity of ether to the gas-oxygen mixture.

Induction, Maintenance and Recovery.

In considering any method of general anæsthesia, our first thought should be the safety and comfort of the patient, our second the most convenient method which will enable the surgeon to carry out the operation to his best advantage. In combining safety with comfort, we must also consider the least harmful and the most pleasant form of anæsthesia for the patient, from the point of view of induction and recovery; for these are the only parts of the anæsthetic state which he can recall, and are the thoughts generally uppermost in his mind when an operation is suggested. If we can assure the patient that the period of induction will be devoid of any unpleasant sensations, that he will be smoothly and rapidly wafted off, and also that the recovery will be just as rapid, we will gain his gratitude and respect. Moreover, should the occasion arise for him to have further operative treatment, he will submit himself much more readily; for the dread and fear of the anæsthetic will have vanished.

Induction.

I am sure that the ideal and most pleasant method of induction of general anæsthesia is with nitrous

oxide. Most patients say they can recollect nothing at all of the induction, a few will recall "a wave of warmth all over" or a "large thick feeling", or perhaps a "tired and numb feeling", but all agree that it was in no way unpleasant.

The period elapsing from the time the mask is put over the face till loss of consciousness ensues is only a matter of a few seconds; this alone is a great boon to the highly nervous patient, who, during a slow induction with ether, for instance, is in deadly fear that the surgeon will begin to operate while he is still conscious. With nitrous oxide he goes under before he has time to think at all.

Maintenance.

Can the surgeon carry out his operation in the manner planned and to his best advantage? Any operation not requiring muscular relaxation can be done under nitrous oxide and oxygen alone. If muscular relaxation is required, there are two alternatives: (i) Ether can be added, about 56 cubic centimetres (two fluid ounces) per hour being required for the average case; (ii) adequate premedication with "Sodium Amytal" or "Avertin" may be used.

Recovery.

In regard to recovery, if nitrous oxide and oxygen are used throughout anæsthesia, the patient wakes up in about five seconds after the mask is lifted from the face. He looks a little dazed, and ten seconds later he will be conscious. This state of affairs holds good if the anæsthetic has been maintained for as long as twenty minutes. If "under" for an hour, the patient seems a little more dazed and apathetic, but soon recovers; he may appear tired and a little flushed, and perhaps vomits once, but does not recollect this. Some plethoric persons, alcoholics, and those people who are "easily upset on the stomach", are more apt to vomit at the close of the anæsthetic, but they rarely vomit more than once or twice.

Premedication.

I consider that premedication has a beneficial effect on the patient before any type of anæsthetic, but as a preliminary measure to nitrous oxide and oxygen it is almost essential if smooth and steady anæsthesia is desired. It brings the patient to the operating theatre in a peaceful state of mind and means an easy and quiet induction. As the recovery from nitrous oxide is very rapid, premedication keeps the patient in a state of analgesia; thus any immediate post-operative pain is avoided.

Premedication is especially valuable in "highly strung" women, and is a necessity when the patients are powerful men, alcoholics, athletes or drug-takers. Morphine alone may be given half to three-quarters of an hour before operation in a dose that would not be regarded as small for the patient's size, age and condition. This dose varies from 0·011 to 0·022 gramme (one-sixth to one-third of a grain). Morphine, as is well known, lowers the general metabolic rate; this enables one to give a lower percentage of oxygen and a higher percentage of nitrous oxide. By this means it is easier to keep the patient in a state of surgical anæsthesia, because the anæsthetic margin is widened. These comparatively large doses of morphine do not cause the same respiratory depression under nitrous oxide and oxygen as they undoubtedly would under chloroform or ether.

Atropine is not added to the morphine, as in some way, not yet fully understood, it upsets the smooth working of nitrous oxide and oxygen anæsthesia. All authorities agree that this does occur. Consequently the administration of atropine is to be avoided, especially to those people who suffer from increased metabolism, such as patients with thyreotoxic goître.

A valuable preliminary is "Omnopon" (also known as "Pantopon"), 0·044 gramme (two-thirds of a grain), and scopolamine, 0·00065 gramme (one one-hundredth of a grain). An ampoule containing this dose is put up by the Hoffmann-La Roche Chemical Company. This is a full dose for a strong patient and must be varied according to the patient's age, weight and habits. Premedication with "Sodium Amytal", 0·01 gramme per kilogram (one grain per stone) weight, given by mouth two hours before the administration of the anæsthetic, makes a very good sedative and brings the patient to the theatre in a dozing, sleepy condition. This dose is well within safe limits.

Paraldehyde in a dose of 0·56 mil per kilogram (one fluid drachm per stone) weight can be given by the rectum, and makes satisfactory premedication, especially for children. It is convenient to mix 3·5 cubic centimetres (one fluid drachm) of paraldehyde with 19 cubic centimetres (half an ounce) of olive oil. This is injected into the rectum after the latter has been washed out. It is given through a catheter and funnel. The patient usually falls quietly to sleep and avoids the preoperative fear so distressing to patients and children alike. It has been used at Great Ormond Street Hospital as an introduction to ether or nitrous oxide anæsthesia with great success and satisfaction. "Avertin" may also be used before nitrous oxide in doses from 50 to 70 milligrammes per kilogram of body weight.

Difficulties of Administration.

There is no doubt that, though nitrous oxide is the most agreeable general anæsthetic to take, it is by far the most difficult to administer successfully. This type of anæsthesia is quite different from ether or chloroform because changes under nitrous oxide occur rapidly, whereas they develop slowly under ether or chloroform, and thus alertness and close attention are essential.

The path of maintenance of smooth and steady anæsthesia is very narrow, and the problem we face is to avoid giving too much oxygen on the one hand and too little on the other. Our first endeavour in the administration of nitrous oxide is to displace the nitrogen, oxygen and other gases

from the lungs, blood and other tissues, and replace them with nitrous oxide. Our second endeavour is to allow our patient just so much oxygen as we find necessary to maintain anæsthesia and prevent cyanosis. If, during maintenance, the patient is given too much oxygen, he will straightway "come out", and if he is not given enough he will develop anoxæmia, showing itself by cyanosis, and leading to asphyxia.

I cannot do better here than quote from a paper by de Coux, showing the meaning of these terms.

> Anoxæmia, cyanosis and asphyxia are not synonymous terms. Anoxæmia, for example, is not the modern word for asphyxia, as one writer naïvely defines it; it merely means the absence of oxygen from the blood. As anoxæmia cannot suddenly be produced, there will be various stages between normally oxygenated blood and anoxæmia, so we speak loosely of various degrees of anoxæmia. The name given to the train of signs caused by depriving the patient of oxygen is asphyxia.
>
> With regard to cyanosis or "blueness", it is possible to have a cyanosed patient who is not being asphyxiated, and *vice versa*. Cyanosis is merely due to there being more hæmoglobin than oxyhæmoglobin in the capillary circulation of the skin. Now in the plethoric patient there is more hæmoglobin than is necessary. In other words, a small percentage of it can carry all the oxygen required for his physiological needs. This being so, the patient can easily be cyanosed without being asphyxiated. In an anæmic patient, on the other hand, there is so little hæmoglobin that all of it is required to carry oxygen in the form of oxyhæmoglobin, and the smallest diminution of oxyhæmoglobin will produce asphyxia, but not cyanosis. (*British Journal of Anæsthesia*, December, 1931).

During nitrous oxide anæsthesia the first sign that the patient needs more oxygen and is threatened with asphyxia is increased respiratory rate. If no oxygen is given to satisfy the patient's needs, inspiration returns to normal, but excessive expiratory efforts become more pronounced, leading rapidly to general clonic and tonic convulsive efforts known as jactitation. In the next stage the pupils dilate and are fixed, all the accessory muscles of respiration are rigidly contracted, and respiration, becoming shallow and sighing, soon ceases. The heart, however, continues to beat after respiration stops, so that if oxygen is given under pressure, the patient soon recovers from this alarming situation.

Utility and Suitability for Particular Operations.

Nitrous oxide and oxygen anæsthesia is very suitable for operative work on diabetic patients; for it causes practically no disturbance of carbohydrate metabolism. Nitrous oxide-oxygen has been shown by Crile to be the anæsthetic of choice for patients suffering from thyreotoxic goître. It is of great value when an operation has to be performed on a patient during the course of a prolonged and debilitating illness. As I have said before, it is a most suitable anæsthetic for those suffering from severe shock and for the repeated dressing of painful wounds.

It is used extensively and successfully in Canada and the United States of America and in England for the relief of pain during labour. For minor operations, such as curettage of the uterus, opening abscesses, cystoscopy *et cetera*, it has many advantages.

Conclusions.

I feel that nitrous oxide is not sufficiently known and used for those operations for which it is most suitable. There are certain drawbacks which prevent the more extensive use of nitrous oxide:

1. Machines and cylinders are heavy and bulky, and are not easily transported.
2. The apparatus and appliances are costly.
3. The cost of administering nitrous oxide and oxygen is between sixteen shillings and one pound per hour.

Reports of Cases.

TUMOUR OF THE MID-BRAIN IN A NEW GUINEA NATIVE.

By H. T. ILLINGWORTH, M.B., Ch.M.,
Formerly Medical Officer, Department of Public Health, Rabaul, New Guinea.

ON November 7, 1930, M., a native of Morobe, New Guinea, entered the Native Hospital, Rabaul, with the following history:

He had definite and increasing weakness of the left side of his body, left leg and left arm. The onset had been gradual, extending over six weeks, and his condition was slowly becoming worse. Nothing else of importance was noted in the history. It is difficult to obtain a history from a native.

On examination dysarthria of a stammering character was found. The patient was unable to walk and could stand only with difficulty and by being supported. His general aspect suggested Parkinsonism, and he gave the impression of being able to understand the questions put to him to a moderate degree, but experienced great difficulty in enunciating words; he drawled his words and left the sentence uncompleted or cut it short. He appeared moderately intelligent, but seemed unable to sustain attention and had a tired, listless expression. His facial muscles lacked animation and gave a definite mask-like aspect. The examination of the alimentary, urinary and respiratory systems revealed nothing abnormal, and his heart sounds and and apex beat were also normal.

There was a slow nystagmus with the quick movement to the left side. The facial muscles had possibly lost some tone, but moved equally and otherwise normally. There was no ptosis, and ocular signs and symptoms were, apart from the nystagmus, negative. The eye movements were unimpaired. No loss of sensation was found in the face. Swallowing was found to be difficult, and coordination of the muscles of mastication and tongue movements was lacking. The tongue was protruded equally on each side, but was tremulous and was not controlled in speech movements.

Babinski's sign was present on the left side; there was hyper-exaggeration of the knee jerks on the same side, with a prolonged ankle clonus and extreme spasticity of the left leg. The ankle jerk was also increased. With the spastic gait was a gross ataxia, which made walking quite impossible.

His left arm, which he was unable to raise above the shoulder, was weak, but the reflexes were unaffected. It was considerably weaker than the right arm, which appeared normal. The right leg, which was weak, was found to be normal in power and reflexes, but lack of tone was evident. The power to coordinate with the other leg in walking movements was quite absent, and it was totally impossible for the patient to make more than one step without immediately losing his balance. He

was even unable to stand with all his weight on his right leg; he had to sit down and then lie down. The left leg had very little power. The lack of control also made it impossible for him to attempt to put any weight on the leg, which had become slightly flexed and was rather flabby from disuse.

No abnormality was detected in the sensory system, but the tests for passive position and passive movement were not satisfactory. The bowels and bladder acted normally. Rombergism was present, also adiadochokinesis. On the left side hypotonia and ataxia were also noted. He was unable satisfactorily to perform the pass point test. He could not touch the tip of the nose rapidly with the forefinger of either hand.

The temperature on admission was 37·8° C. (100° F.) and the pulse rate 80 per minute.

The following were suggested as possible causes of the condition: (i) Cerebral malaria, (ii) frambœsia, (iii) tuberculous meningitis, (iv) *encephalitis lethargica*, (v) cerebral tumour.

On November 8, 1930, a specimen of blood-stained cerebro-spinal fluid was obtained. On November 10, 1930, by a further lumbar puncture, sixty cubic centimetres (two fluid ounces) of blood-stained fluid, not under pressure, were obtained. On November 11, 1930, a further quantity of fluid was obtained; attempts to culture organisms from it failed. The leucocytes in the cerebro-spinal fluid were in normal proportion to the red blood cells, and there was no increase in the protein content. In other respects it was normal. A leucocyte count was found to be normal; examination of the fæces revealed the presence of hook-worm; no malarial parasites were isolated from the blood. A course of anti-frambœsial treatment was given over a period of ten days. The absence of dramatic improvement from this measure, combined with a failure to respond to quinine therapy and the absence of any evidence of tuberculosis in the cerebro-spinal fluid, narrowed down the diagnosis to *encephalitis lethargica* or cerebral tumour.

On November 20, 1930, some slight improvement in the patient's general demeanour was noticed, but the definite signs found remained constant. He improved by gaining some confidence and tried with a little more success to stand up, but this was never done without assistance.

These were all the evidence to be obtained. The patient continued in his same state till about February 15, 1931, when he became entirely bedridden, lost his appetite, commenced to lose weight and steadily went down hill. He died on February 22, 1931.

The *post mortem* examination revealed generalized abdominal tuberculosis and a tumour of the mid-brain. The other systems were clear.

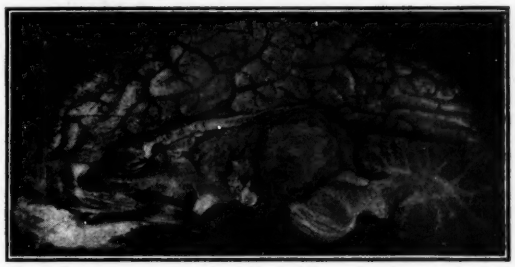
Dr. T. H. R. Mathewson has made a macroscopical survey of the exact anatomical site of the tumour, and Dr. Duhig has examined the growth histologically.

The temperature varied the whole time between 36·1° and 38·9° C. (97° and 102° F.) and the pulse rate between 95 and 110 per minute. During the final week the temperature was consistently below normal and the pulse rate very close to 100 per minute.

I have no access to the literature on this class of tumour, but I believe it is very rare.

Report on the Macroscopical Appearances

The brain submitted for examination and description has been sectioned about one centimetre to the right of the mid-line. In the section the tumour can be seen situated in the posterior part of the optic thalamus. It is circular in outline and in it is a smooth central greyish area, over which are dotted areas of a darker grey colour. This central area is firm in consistence and is bordered superiorly, posteriorly and inferiorly by a margin of pale yellow soft material in which a few small cavities appear. Surrounding the whole tumour is a narrow margin of dark grey material. The central area measures 2·5 centimetres in diameter and the yellow margin one centimetre at its widest part posteriorly. In its upper part the tumour is separated from the lateral ventricle by the narrow grey margin and, above this, a still narrower band, which together measure 0·5 centimetre in width. The posterior third of the *corpus callosum*



The right half of the brain, showing the tumour situated in the mid-brain, involving the optic thalamus, *corpora quadrigemina*, *pons Varolii* and superior cerebellar peduncle.

which lies over the tumour, is thinned out, and towards the splenium tapers into a leaf-like structure, not more than 0·25 centimetre thick. Anteriorly the tumour is bounded by the anterior part of the optic thalamus; posteriorly it extends to within one centimetre of its posterior extremity. Inferiorly the tumour is bounded by the pons, and, further forward, it comes to within a distance of 0·5 centimetre of the surface of the brain forming the roof of the interpeduncular space. The tumour extends outwards as far as the lateral extremity of the optic thalamus.

Histological Report.

The tumour was submitted for histological examination to Dr. J. V. Duhig, who reports that the tumour is caseous and is teeming with tubercle bacilli. The organism was both acid and alcohol fast, and the decolorizing acid was of sufficient strength to decolorize leprosy bacilli. The growth is a tuberculoma.

Reviews.

HUMAN HEREDITY.

"GENETIC PRINCIPLES IN MEDICINE AND SOCIAL SCIENCE", which has been written by Lancelot Hogben, the newly appointed Professor of Social Biology in the University of London,[1] will administer a slight "jar" to those medical men who still have no knowledge of the scientific methods of investigating heredity and, on the other hand, a tepid if not a cold douche to those kindly folk who have done much to discredit eugenics by their blind belief in an impotent environment.

There exist today many excellent books on the mechanism of heredity, on genetics and animal breeding, and one or two others more specially devoted to human inheritance. Hogben does not attempt to describe the mechanism of inheritance or catalogue the data of human heredity, but sets out to provide an indication of the methods by which the "materials of human pathology may be brought within the scope of a quantitative treatment consonant with the principles of animal breeding".

There is a very considerable application of mathematics in the most recent researches in this field, and with the exception of the last chapter, which is more speculative, the author sums this work up in an able manner and in the cold light of facts. A knowledge of the elements of Mendelian heredity is assumed to be possessed by the reader.

In the first chapter the keynote is struck by taking up the problem of the resemblance of twins and subjecting the facts to an analysis in order to measure the relative

[1] "Genetic Principles in Medicine and Social Science", by Lancelot Hogben, M.A., D.Sc.; 1931. London: Williams and Norgate. Demy 8vo., pp. 230. Price: 15s. net.

effects of heredity and environment. Following on this the author deals with human characters which are undoubtedly due to simple Mendelian factors in heredity, characters such as colour blindness, hæmophilia, Leber's disease and night blindness. Criticism of some of the older interpretations of family pedigrees is made, and it is pointed out that although family pedigrees provide data which conform to the quantitative requirements of modern genetic interpretation, there is ground for belief that the importance of uterine environment as a factor which may simulate the influence of heredity in the determination of congenital conditions, has been too much neglected. Chapter III, one of the most interesting, deals with the utilization of the discoveries of the "blood groups" in the investigation of the mechanism of human heredity. Chapter IV is devoted to the genetic basis of social behaviour and the inheritance of mental deficiency. Here again the author is very careful to distinguish between heredity and the possible effects of environment. The futility of discussing "feeble-mindedness" as if it were a simple clinical entity is emphasized. The study of juvenile amaurotic idiocy demonstrates more or less clearly a disorder due to a genetic basis; the study of other mental disorders suggests environmental agencies in which are included size of family, prenatal nutrition, toxæmias of pregnancy *et cetera*.

After discussing the concept of race, the phenomena of race-crossing, and the growth of human population, the author proceeds to give some of his own opinions on the social application of genetic principles. With much of this we are inclined to agree, but it is a pity that in a book of such value, which sets out to keep so strictly to facts, the author should let some obvious personal feeling escape him. He could have made all his points without descending to this.

The book can be recommended to all who are interested in the study of human heredity, and particularly to those who are interested in pathology, mental deficiency, social reform or eugenics.

RHEUMATIC TREATMENT AT BATH.

"RHEUMATOID ARTHRITIS AND ITS TREATMENT" is a short brochure by Vincent Coates and Leo Delicati, in which the authors describe the methods of treatment as used by them at the Royal Mineral Water Hospital, Bath.[1] The authors reserve the term rheumatoid arthritis for a subgroup of cases under the heading of infective arthritis. They describe their findings in relation to one hundred consecutive patients treated by them at the Royal Mineral Water Hospital.

Stress is laid on the prodromal symptoms of nervous exhaustion, and the authors point out the difficulties in the differential diagnosis. Attention is drawn to the association of thyreoid dysfunction with rheumatoid arthritis in 20% of the cases in the series.

The authors also point out the not uncommon occurrence of rheumatoid arthritis following immediately after true rheumatism or at a later period. Rheumatic heart lesions occurred in 4% of the patients, which is much above the incidence of this disease among the general population. They advise a full blood count, the performance of a sedimentation rate test and the giving of a fractional test meal in every case. Great reliance is placed on the diagnostic value of an increase in the sedimentation rate. This occurred in 97% of the cases. It is of especial assistance in deciding the acuity of the disease when the patient appears apparently well and increased exercise is being considered, and it is also of great value in differential diagnosis from gout or endocrine arthritis.

Treatment is aimed at increasing the patient's immunity by general methods, such as rest, sedative baths, the administration of thyreoid extract, diet, "remineralization" and vaccines. Vaccines are not used as a routine in the Royal Mineral Water Hospital at Bath, but the authors are willing to believe that they do good in certain cases.

Attempts at stamping out the infection are made by intestinal disinfection, diuretics, and increased action of the skin by special baths.

The special treatment of the joints is mainly directed at lessening the resulting deformity and contractures. Due weight is given to the necessity for splinting and for active and passive movement to free the minute adhesions in the affected joint cavities, which, if not broken down, become later organized into fibrous adhesions with marked restriction of movement.

The authors describe particular splints for individual joints, and also hold that the patient should exercise in a specially constructed large bath that supports his limbs and can be kept at an even temperature.

The latter part of the book is devoted to the description of cases of different types of the disease.

The whole is a well written and pleasing account of some aspects of the subject. It is in no way deeply scientific; in fact the question of ætiology is not touched at all. The book could not be recommended as a text book on the subject.

[1] "Rheumatoid Arthritis and Its Treatment", by V. Coates, M.A., M.D., M.R.C.P., and L. Delicati, L.M.S.S.A.; 1931. London: H. K. Lewis. Crown 8vo., pp. 128, with illustrations. **Price: 6s. net.**

PSYCHOLOGY.

PROFESSOR WOODWORTH is to be congratulated on writing a survey of his specialty in a recent book, "Contemporary Schools of Psychology".[1] The multiplicity of apparently contradictory theories has serious disadvantages, creating as it does an impression that if the experts cannot agree, why need the man in the street worry? Professor Woodworth deals adroitly with this aspect; he shows the utility of research on many lines, and whilst he will not prophesy, he hints that the day of the final battle is not far distant. Each will have been victorious by contributing positive facts of great importance. His chapter headings indicate the subject matter: "Introspective Psychology and the Existential School", "Behaviourism", "Gestalt Psychology or Configurationism", "Psycho-Analysis and Related Schools", "Purposivism or Hormic Psychology". His last chapter is entitled the "Middle of the Road". Supposing that a world tournament of two or three thousand psychologists were organized on a large field with banners raised here and there as rallying points for the adherents of the schools; there would remain a large body in the middle of the field ready to watch the jousting. The body of the unattached would form the majority. It is comforting to note his view that it does not consist simply of the multitude, the little fellows.

The author strongly defends the attitude of sitting on the fence. Whilst the extremists make men of straw on which to test their weapons, the passive observer is able to pick up useful fragments. Gradually a wearing down process is taking place and material from the sides is finding its way into the middle to become the general property. To a certain extent this is to be regretted, because the present warfare is exciting and picturesque. Harmony will be humdrum and prosaic.

The aim of the book is to give an impartial view of modern trends. Professor Woodworth has undoubtedly succeeded in his task. His bibliography is large, and whilst many well known names are missing, space would obviously not permit the mention of all. His transparent sincerity and capacity for appreciating good work, in whatever guise, should serve as a model of the true scientific outlook. The book leaves pleasant thoughts in the mind of the reader. Psychology is a subject, great, colourful and vivid, created by men possessing the attributes of greatness. Moreover, Professor Woodworth writes in an interesting fashion.

Enough has been said to indicate that this is a book which is worthy of a place on the bookshelf of all who are interested in psychology, and will further enhance the author's reputation.

[1] "Contemporary Schools of Psychology", by R. S. Woodworth, Ph.D., Sc.D.; 1931. London: Methuen and Company Limited. Crown 8vo., pp. 255. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 4, 1932.

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THE POLIOMYELITIS PROBLEM.

POLIOMYELITIS presents a difficult problem. It would appear at first sight that a disease characterized by obvious paralysis and occurring more often than not in epidemics, would present no great difficulties either in diagnosis or treatment. To the uninitiated the paralysis would establish the diagnosis without much possibility of error and the uninitiated would also reduce treatment, once the acute stage of the disease had passed, to massage and the use of electricity and of other forms of physical therapy, so that the paralysed muscles might be "strengthened". The truth of the matter is that neither diagnosis nor treatment is so simple as the uninitiated would think. Both diagnosis and treatment must be based on a proper conception of the disease as an infective process characterized by definite preparalytic and paralytic stages, on the physiological condition of muscles which are deprived of their nerve supply, and on the results which may be expected to follow different therapeutic measures. All these facts have been set out over and over again. Discussions have been held on the subject at British Medical Association meetings, post-graduate courses have been given, clinical demonstrations have been held, special articles have been printed in medical journals both on diagnosis and treatment, and yet the problem remains. That these efforts have borne some fruit is shown by Dr. Steigrad, whose article, based on observations made at the Royal Alexandra Hospital for Children, is published in this issue. Dr. Steigrad points out that every one of the ninety-three patients treated at the Royal Alexandra Hospital in the 1929 epidemic manifested some form of paralysis. On the other hand, of the patients admitted in the present season, in whom a diagnosis of anterior poliomyelitis was definitely established, 30% had no paralysis or paresis on admission. The poliomyelitis problem consists, not in the solution of the inherent difficulties of diagnosis or treatment, but in making every medical practitioner realize how he may diagnose the condition and what steps he should take to treat persons affected by it. Dr. Steigrad's statements show that it is reasonable to hope for a solution of the problem on these lines.

The question of diagnosis will merely be mentioned in passing. It was discussed in these pages only a few weeks ago, and has been exhaustively dealt with in special articles. In spite of all that has been written, there are continually being brought to the notice of experts cases in which an obvious diagnosis has been missed, even when paralysis has occurred and when other poliomyelitic infections have been reported in the neighbourhood of patient and practitioner. That such things can happen is astounding. The only remedy to be suggested is that medical practitioners should seek the advice and help of others when they are in doubt. The results of mistakes are so calamitous to the patient and add such a burden to an already overburdened community that every effort must be made to avoid them, even if diagnostic pride and personal reputation are to suffer.

The responsibilities of medical practitioners do not end when a diagnosis is made. Irreparable damage is being done to many sufferers by improper treatment. In these circumstances either the medical attendant does not know the extent to which rest should be imposed on poliomyelitic patients or he is led away by the popular belief that paralysis always calls for massage, and allows his better judgement to be overcome by a wish to

satisfy importunate parents. The use of improper and harmful treatment is causing much concern to those who are in a position to know the facts. The article by Dr. E. B. M. Vance, appearing in this issue, is addressed to medical practitioners who are attending patients convalescent from anterior poliomyelitis. It bears the *imprimatur* of no less an authority than Sir Charles Clubbe, who came to the office of this journal to discuss means of bringing it to the notice of medical practitioners. There are three statements in Dr. Vance's paper on which special emphasis should be laid. The first is that generally two years will elapse before the maximum recovery of a paralysed muscle will take place. The second is that the principles involved in adequate treatment are prolonged muscle rest in splints, interrupted only by daily muscle reeducation exercises. The third is that nothing in the world can lead a weak muscle back to strength except use of the muscle at the bidding of the will.

To state the facts is an easy matter; to have them assimilated by every medical practitioner is much more difficult. We would suggest that those who read these words might discuss them when they talk "shop" with their *confrères*. They might also bring forward the subject of after-care in poliomyelitis for discussion at meetings of local associations and clinical societies. Only if interest is aroused will an impression be made. The urgency of the problem, as we have stated it, makes it the duty of every interested practitioner to awaken his neighbour from slumber.

Current Comment.

TUBERCULIN TESTS.

THE various tuberculin tests have been subjected to a vast amount of investigation. It might almost be legitimate to say "too much investigation"; for, in the continuous argument spread over thousands of pages in practically all the medical journals of the world, there has been repetition almost *ad nauseam*, and medical practitioners have grown weary. They have tired of a controversy that brings them no nearer an appreciation of the clinical value (if any) of tuberculin tests. For this and other reasons all the tests have been practically discarded by great numbers of medical practitioners. This is unfortunate; for there must be a sphere in which tuberculin tests can be usefully employed. The question has been previously discussed in these pages; recently, in the issue of August 8, 1931, and again by Dr. Cotter Harvey in a special article on aids to diagnosis in the issue of November 14, 1931. Dr. Harvey gives a concise expression of modern medical opinion that should be of considerable value to those seeking knowledge concerning the clinical applicability of the tests of sensitivity to tuberculin. The reason for the present reference to the subject is the publication, by the Medical Research Council of the Privy Council, of the report of an investigation by P. D'Arcy Hart.[1] The report merits the earnest consideration of every medical practitioner who comes in contact with tuberculosis, especially tuberculosis of children.

Hart investigated 1,030 persons of all ages whose condition had been diagnosed clinically, beyond all reasonable doubt, as tuberculous, and 751 persons of all ages who were clinically non-tuberculous. As a result of his investigations he concludes that the intracutaneous (Mantoux) test is preferable to the von Pirquet test on the grounds that it is more sensitive, that it can be standardized, and that it permits of accurate dosage. He quotes Opie's remark that the Mantoux test is "the only means by which sensitiveness to tuberculin can be measured so accurately that two successive tests can be compared. The subcutaneous test (the *stichreaktion* of Escherich) is allied to the intracutaneous test. Hamburger believes that the *stichreaktion* has great advantages over the von Pirquet test from the point of view of sensitiveness. Hart can see no advantages in it over the Mantoux test, providing the latter is properly performed.

Hart's method of carrying out the intracutaneous tuberculin test is roughly as follows. Human old tuberculin is used in every instance; there is apparently no qualitative difference in the reaction of animals and man to the human and bovine types of tuberculin. Dilutions of 1 in 10,000, 1 in 1,000, 1 in 100, and 1 in 10 are made. An initial dose of 0·1 cubic centimetre of the tuberculin in the weakest dilution is injected. If this is followed by no reaction, 0·1 cubic centimetre of tuberculin diluted to 1 in 1,000 is employed, and so on, until, if there is no reaction when the weaker dilutions are used, 0·1 cubic centimetre of tuberculin diluted to 1 in 10 is given. Retesting should be carried out within a week of the previous test, or not for several months, on account of the danger of sensitization and consequent severe reaction. Results should be read forty-eight hours and ninety-six hours after the injection, or, if only one observation is practicable, seventy-two hours. Non-specific reactions sometimes occur; but they usually reach their maximum within twenty-four hours and have disappeared by the end of forty-eight hours, when the specific reaction is most pronounced. In some instances, however, when the stronger dilutions are used, non-specific reactions may persist for several

[1] "The Value of Tuberculin Tests in Man, with Special Reference to the Intracutaneous Test", by P. D'Arcy Hart. Medical Research Council of the Privy Council, Special Report Series, Number 164.

days. Confusion may be avoided by the use of glycerinated broth control injections whenever a dilution stronger than one in one hundred is employed. Hart believes that control injections are necessary only in such instances. The possible occurrence of non-specific reactions is the reason for discouraging the reading of the results of the test earlier than forty-eight hours after the injection. The development of an area of erythema, five millimetres in its widest diameter, is regarded as proof of a specific reaction.

There is no need to reiterate that a reaction to a tuberculin test in adult life merely indicates that infection with the tubercle bacillus at some time has taken place. On the other hand, a reaction in infancy should give rise to a strong suspicion that the child is suffering from active tuberculous disease. A high proportion of infants who react to the Mantoux test later develop clinically recognizable tuberculosis, and many die of the disease during childhood. It is worthy of note, however, that the investigators quoted by Hart in support of this view dealt almost entirely with contacts, hospital patients, children in poor circumstances, and children who had symptoms suggestive of tuberculosis when they were tested. Repeated tests and prolonged observation of large numbers of unselected persons in all sections of the community would be necessary for the proof or disproof of the contention. Hart expresses the opinion that, unless there is clear bacteriological or pathological proof, a diagnosis of tuberculosis in childhood should never be made without the evidence provided by a tuberculin test.

It is unfortunate that in the past few years medical practitioners have lost faith in the use of tuberculin tests in the exclusion of tuberculosis; for it is just in this field that the Mantoux test has its greatest value. In Hart's investigations, only 2% of all patients suffering from tuberculosis failed to react. The absence of a reaction must be taken, therefore, as very strong evidence of the absence of tuberculous infection. Hart found that persons suffering from very advanced phthisis often did not react when a weak dilution of tuberculin was used, but that only 1% failed to react when a dilution of one in ten was employed. Contrary to the findings of many investigators, he observed that, though persons suffering from pulmonary tuberculosis associated with profound toxæmia lost their sensitiveness to tuberculin a few days before death, those suffering from miliary tuberculosis and tuberculous meningitis remained sensitive to the end.

Hart advances evidence to show that persons actually suffering from tuberculosis react more readily to tuberculin in weaker dilutions than do persons who have withstood an invasion by tubercle bacilli and are not affected with tuberculous disease. This suggests that, if a sufficiently weak dilution were employed, tuberculous persons only would react; unfortunately, however, such a dilution would need to be so weak that very few reactions would be elicited, and the test would not be of great practical value, save in the diagnosis of tuberculous diseases of the skin, in which conditions sensitivity is often very pronounced. It is only when weak dilutions are used that a reaction in a person over five years of age is of any value in the diagnosis of tuberculosis. The interpretation of the reactions in such cases requires considerable experience, and should not yet be attempted in routine practice. Hart believes, however, that some method depending upon reaction or absence of reaction to injections of tuberculin in varying dilutions may eventually be evolved.

He stresses the value of tuberculin tests in the diagnosis of *lupus pernio* and the sarcoids. Though these conditions are believed to be tuberculous, they are associated with loss of sensitivity to tuberculin; a reaction is evidence against the diagnosis of *lupus pernio* or sarcoid.

A further interesting feature of Hart's report is the suggestion that the systematic performance of tuberculin tests may reveal information concerning a new clinical entity, namely, the transient and benign illness that accompanies an initial tuberculous infection. No doubt the nature of this illness usually escapes recognition. The development of sensitivity by a child who formerly did not react, may assist the medical practitioner to make a correct diagnosis.

There is much else of value in the report, including important observations on the epidemiology of tuberculosis. It should be read by all to whom it is available. Hart has shown that there are great possibilities yet in the proper employment of tuberculin tests. The medical practitioner is well advised to be cautious in his interpretation of reactions to these tests, but he should realize that they are of definite value and may become of far greater value. A point that must be stressed is that a standardized tuberculin must be used if uniform results are to be obtained.

ANÆMIA AND THE GASTRIC JUICE.

IN the prevention of anæmia the healthy gastric juice apparently plays an important part. A recognition of this has induced many investigators to study the problem. Castle and his associates have produced evidence to show that the gastric juice in pernicious anæmia is lacking in some vital factor. Roger S. Morris, Leon Schiff, George Burger and James E. Sherman have recently published a preliminary report on their investigations of the effects produced by the intramuscular injection of normal gastric juice.[1] They found that the administration of gastric juice that had been rapidly concentrated *in vacuo*, effected a rapid pronounced hæmatopoietic response. They believe that gastric juice contains a specific hæmatopoietic hormone, to which they propose to apply the name "addisin", after Thomas Addison. Their views may or may not be correct; but they have discovered what may prove to be a potent therapeutic agent.

[1] *The Journal of the American Medical Association*, March 26, 1932.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

The Rate of Flow of the Cerebro-Spinal Fluid.

H. NICHOLSON (*American Journal of Physiology*, February, 1932) finds that in dogs anæsthetized with morphine and urethane the inhalation of gas mixtures containing 10% to 14% of carbon dioxide and 21% to 26% of oxygen resulted in a marked increase in the rate of flow of the cerebro-spinal fluid from cannulæ placed either in the spinal subarachnoid space or in the *cisterna magna*. The increase occurred immediately upon the administration of the gas, rose to a maximum after several minutes and then either maintained a high level until the administration ceased, or, more rarely, fell gradually while the high concentration of carbon dioxide was still being inspired. In any case, the increased rate of flow continued throughout the period of administration of the gas mixture, even if this were prolonged to twenty-five minutes. When room air was inspired, the rate of flow fell rapidly to the normal level, and in some cases even below this level. Low oxygen tensions caused more complex effects. Inhalation of a gas containing 6% of oxygen almost invariably resulted in a rapid and marked decrease in the flow. If the administration of the oxygen-poor gas mixture was sufficiently prolonged, the primary decrease was followed by an increased rate of flow up to, or even exceeding, the normal. Still further prolongation of the administration caused little change in the flow. In most cases the readmission of room air was followed by a rapid decrease and a subsequent increase of flow to approximately the rate maintained during the breathing of the gas poor in oxygen. In other cases there was an initial rise followed by a fall, while in a few the restoration of normal alveolar oxygen tensions seemed to have no effect. Artificial ventilation of the lungs with gas containing high percentages of carbon dioxide produced the same effects as normal respiratory ventilation. The effects of artificial administration of oxygen-poor mixtures differed from those resulting from normal ventilation with similar mixtures. A prompt and pronounced increase in the rate of flow invariably occurred. If the administration was sufficiently prolonged, a slight decrease from the maximum level might occur, but in general the increase was maintained until the oxygen in the inspired air was restored to normal. To exclude any influence of respiratory movements on the escape of cerebro-spinal fluid, the observations on the effects of artificial ventilation were repeated in curarized animals and the same results were obtained. Simultaneous observations on the systemic blood pressure appeared to exclude vascular changes as the cause of the variations in the flow of cerebro-spinal fluid. The author concludes that the observed results indicate variations in the rate of formation of the cerebro-spinal fluid, and suggests that these are the result of changes in blood reaction. It is considered that an increased acidity of the blood increases the activity of the chorioid plexuses, and, conversely, a diminished rate of cerebro-spinal fluid formation results from an increase in the alkalinity of the blood.

Periodic Ventilation Induced by Exposure to High Pressures of Oxygen.

J. W. BEAN (*American Journal of Physiology*, March, 1932) records the occurrence of periodic respiration in dogs as a result of exposure to oxygen at a pressure of approximately five atmospheres. Very long apnœic periods, up to twenty minutes in length, were frequently observed. During these periods the colour of the blood changed from bright arterial to a very dark venous hue, indicating that the residual oxygen in the lungs had been exhausted. Complete cessation of the cardiac contractions for as long as forty-five seconds was sometimes observed during these periods of suspended breathing. There was as a result a fall in blood pressure to zero. In some cases this failure in the cardiovascular system was accompanied by the restoration of the respiratory movements. As the heart beat was restored and the blood pressure rose, a further period of apnœa supervened; this alternation might be repeated several times. In general this type of periodicity progressed to a failure of the respiratory and cardiovascular systems and death. A second type of periodicity was not accompanied by apnœic periods; but phases of increased and decreased pulmonary ventilation occurred simultaneously with augmentation and diminution in cardiac rate and systemic blood pressure. A combination of these types of periodic breathing might occur in the same animal. Improvement in the general condition accompanied the onset of the second type of respiration. The periodicity might occur at the height of exposure to the increased oxygen tensions and subside on decompression, or it might occur only during decompression or after the pressure had fallen to one atmosphere of oxygen.

The Relation of Prolan to the Anterior Hypophyseal Hormones.

OBSERVATIONS by H. M. Evans, K. Meyer and Miriam E. Simpson (*American Journal of Physiology*, March, 1932) indicate an interesting relationship between the substance excreted in the urine of pregnant women which is responsible for the Aschheim-Zondek test, and the gonad-stimulating and growth-promoting hormones of the anterior pituitary lobe. In the absence of the pituitary gland, the substance which in the normal infantile rodent causes the increase in size and vascularity of the ovarian follicles on which the Aschheim-Zondek reaction depends, no longer produces its characteristic effects. This substance, prolan, has a limited effect on the ovaries of the normal infantile rat. Increase in dose is accompanied by a corresponding increase in the weight of the ovaries up to a certain maximum. Further increase in the dose of prolan is not followed by any increased effect on the ovary. On the other hand, the administration of the gonad-stimulating hormone from the anterior lobe of the pituitary to the rat whose pituitary gland has been removed, causes full development of the genital system, and in the infantile animal the administration of this hormone in increasing doses results in a continued effect on ovarian weight to a value greatly in excess of the maximum weight resulting from the injection of prolan. If prolan is combined with a pituitary extract, which is itself not capable of producing sexual maturity, an increase in ovarian weight comparable with that produced by a potent gonadotropic pituitary extract often occurs. The authors suggest that the gonadotropic hormone is present in the hypophysis in an inactive state and only exerts its effects after it has been in contact with an activator. This activator is the substance which is known as prolan and which is found in greatest concentration in the urine of pregnant women. It may be present in the pituitary gland, both in the adult and infantile animal. In the normal infantile animal the effect of prolan is limited by the amount of gonadotropic hormone available. The addition of more of the inactive precursor of this hormone enables increased doses of prolan to exert effects on the ovary in excess of the maximum attained by the injection of prolan alone. Even extracts of the growth hormone of the anterior lobe of the pituitary, which are completely free from any effects on the ovaries, assume gonadotropic actions when combined with prolan. On the other hand, preparations of the gonad-stimulating hormone free from growth hormone cannot be further activated by prolan. It is suggested that the growth hormone may be the precursor of the gonadotropic factor. Any treatment of the extracts which destroys the growth-promoting function also results in a failure of the activation on subsequent combination with prolan.

The Relationship of Heart Size and Body Build to Cardiovascular Efficiency.

ACCORDING to H. A. Treadgold and H. L. Burton (*The Lancet*, February 6, 1932), the apex beat of the heart, as determined by palpation, coincides with the anatomical position of the apex or the left border of the heart as found by the orthodiagram in two-thirds of cases. The error in the remaining cases rarely exceeds one centimetre. In their series of 223 cases the total transverse diameter of the heart is within 10% of the normal

predicted from weight alone in 87%. In persons of average build 90% of hearts fall within normal limits, 6% having transverse diameters below and 4% above normal. Of people of slender build 12% have transverse cardiac diameters more than 10% below normal limits, and of thick-set persons 11% have hearts with transverse diameters more than 10% above normal. Among cases of cardiovascular inefficiency as judged by Royal Air Force tests, a high percentage (23%) of abnormally small hearts was found. Only 4% of persons reaching a satisfactory standard of efficiency had such small hearts. Cardiovascular inefficiency is most common in those of slender build.

BIOLOGICAL CHEMISTRY.

The Calcium, Protein and Inorganic Phosphorus of the Serum of Children.

GENEVIEVE STEARNS AND G. CLINTON KNOWLTON (*Journal of Biological Chemistry*, August, 1931) have determined the calcium, protein and inorganic phosphorus content in 76 sera from children ranging in age from earliest infancy to sixteen years. The blood was taken from the arm or jugular vein in the older children, from the longitudinal sinus in infants, and from the cord vein in the newly born. The methods used for the determinations are given. All of the analyses were made during the summer months. The children were given cod liver oil daily and were out of doors during the major part of the day. No data from nephritic or cardiac patients or from patients with obvious disturbance of calcium metabolism were included in the series. No constant relationship was observed by these workers between the level of serum calcium and serum protein or serum inorganic phosphorus; these findings are in direct contrast to the findings in nephritis in childhood.

Some Effects of a Ketogenic Diet.

RICHARD W. B. ELLIS (*Archives of Diseases in Childhood*, October, 1931) has investigated the tolerance of children between the ages of three and eight years to a high fat diet in various conditions. The effect of ketosis on post-anæsthetic vomiting was first studied. A comparison was made of two groups, each of seven children, who were subjected to tonsillectomy. The first group was prepared before operation by the administration of large amounts of carbohydrate; in the second group no food was given on the day of the operation. All the children in the second group manifested considerable ketosis, while in the first group it was slight or absent. In only one case was there undue vomiting, and this was in a child in the second group. It appeared, therefore, that a considerable post-anæsthetic ketosis could exist without necessarily giving rise to vomiting. Artificially produced ketosis before and after operation was found to be well tolerated and not to increase the vomiting or to cause other symptoms. The effect of a ketogenic diet in two typical cases of cyclical vomiting was studied when the patients were free from symptoms. Both children rapidly developed and maintained a marked ketosis, but in neither was there any nausea, vomiting or other evidence of intolerance. The intolerance to carbohydrate produced by a high ketogenic diet which has been shown to exist by previous workers, was investigated by means of the glucose tolerance test, and it was found in each case investigated that ketosis was associated with a reduced carbohydrate tolerance. Insulin tended to readjust the sugar curve to normal, but sodium bicarbonate had no effect in lowering the intolerance. The author does not claim that ketosis can never be responsible for clinical disturbances, since in extreme degrees of ketosis considerable change in acid-base equilibrium may take place. He does suggest, however, that a considerable ketosis may be well tolerated by many children, including some who are subject to cyclical vomiting, and normal children undergoing operation. He believes that the diagnosis of ketosis as an explanation of symptoms should be made with the greatest caution and when every other possible cause has been excluded.

The Acetone Content of the Blood in Phlorizin and Pancreatic Diabetes.

HAROLD E. HIMWICH, W. GOLDFARB AND A. WELLER (*Journal of Biological Chemistry*, October, 1931) have attempted to determine the effects of various organs on the acetone content of the blood, by analysis of samples of the afferent and efferent blood for acetone bodies. The substances investigated were liver, muscle, kidney and the organs drained by the portal vein. Sixty-four observations were obtained on six dogs treated with phlorizin, and seventy-six on twelve dogs from which the pancreas had been removed. The liver was found to be the chief site of acetone production in twenty-seven out of twenty-nine experiments. Muscle and the organs drained by the portal vein liberated acetone bodies in eighteen experiments and removed acetone in twenty-four others. The author reviews previous work done on this subject and believes that his work indicates that the acetone excretion by the kidneys and lungs is the algebraic sum of the actions of the various organs.

Diffusibility of the Proteins of Normal and Pathological Plasma.

OLIVER HENRY GAEBLER (*Journal of Biological Chemistry*, October, 1931) has carried out a series of simple dialysis experiments with blood plasma from normal subjects and from patients with and without albuminuria. He sought to determine whether there is present in nephritic blood any substance which increases the permeability of collodion membranes to protein, lowers the surface tension of the blood, and is a factor in the production of the albuminuria as postulated by Clausen. Flat collodion membranes of high permeability were found to show a different permeability for proteins in opposite directions. If the side of the membrane which was uppermost during the evaporation stage of preparation was placed toward the protein solution, less protein passed through than if the side which was towards the plate was placed toward the protein. Alcohol-treated membranes, prepared according to the technique of Nelson and Morgan, were found very suitable for dialysis experiments with proteins. The diffusibility of the proteins of plasma of normal persons was considerably greater than that of any plasma obtained from patients. The results from the cases of chronic nephritis were practically the same as those obtained from a second group of patients from whom proteinuria was absent. Contrasting sharply with these results are the results obtained from four specimens of plasma in two cases of nephrosis. Here the diffusibility was found to be considerably reduced. A sufficient number of cases was not studied to determine whether the observed changes in the diffusibility were referable to changes in the proteins or to the presence of other substances in the plasma which are taken up by the membrane. The author concludes that these dialysis experiments with the membranes used yield no evidence that proteinuria is due to an increase in the diffusibility of plasma proteins or to the presence of substances increasing the permeability of artificial membranes.

The Liberation of Glucose by Adrenalin.

JESSE L. BOLLMAN, FRANK C. MANN AND CHARLES M. WILHELMJ (*Journal of Biological Chemistry*, September, 1931) have undertaken a series of experiments, the results of which reveal that adrenalin causes a pronounced decrease in the glycogen content of the muscles of depancreatized dogs. The amount of extra glucose that appeared in the urine, corresponded closely to the amount of glycogen lost from the muscles. This glycogen lost from the muscles is regarded as the source of glucose lost under these conditions, and no evidence was obtained of any additional excretion of glucose that could have been derived from fatty acids or other material. Data were also obtained to show that the action of adrenalin was similar in normal dogs. Several hours after an injection the glycogen content of the liver was not materially changed, but the glycogen content of the muscles was greatly decreased. If glucose was given with the adrenalin the glycogen content of the liver generally increased, but the glycogen content of the muscles decreased. In hepatectomized animals there was no definite decrease of the glycogen content of the muscle after adrenalin nor was there any increase of the lactic acid content of the blood or urine, such as occurs in the normal dog. If glucose as well as adrenalin be administered to the hepatectomized animal, the glycogen content of the muscles may increase.

British Medical Association News.

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association has forwarded the following list of books that have been added to the library of the New South Wales Branch.

"The Work of the Digestive Glands: Lectures by Professor J. P. Pawlow", translated by W. H. Thompson; "Recent Advances in the Physiology of Digestion: Mercers' Company Lectures", by Ernest H. Starling; "Transactions Eighth Congress, Far Eastern Association of Tropical Medicine"; "Those Teeth of Yours", by J. M. Campbell (William Heinemann); "Cancer and Scientific Research", by B. Holmes (The Sheldon Press); "Practical Radiation Therapy", by I. I. Kaplan (W. B. Saunders); "International Studies, Volume III", by A. Newsholme (G. Allen and Unwin); "Hay Fever, Hay Asthma, etc.", by W. Lloyd (Straker Brothers, Limited); "General Surgery", by E. A. Graham (Year Book Publishers); "Midwifery by Ten Teachers", edited by C. Berkeley, Fairbairn and White (Edward Arnold); "Emergency Surgery", Volume II, by H. Bailey (John Wright and Sons); "Recent Advances in Radiology", by P. Kerley (J. and A. Churchill); "Traumatotherapy", by J. J. Moorhead (W. B. Saunders); "Physical and Radiological Examination of the Lungs", by J. Crocket (H. K. Lewis); "Manual of General Practice", by W. S. Sykes (H. K. Lewis); "History of Medicine", by B. Dawson (H. K. Lewis); "Ultra-Violet Therapy", by A. Furniss (William Heinemann); "Introduction to Practical Bacteriology", by T. J. Mackie and J. E. McCartney (E. and S. Livingstone); "Gastric Acidity", by J. D. Robertson (John Murray); "Wheeler and Jack's Handbook of Medicine", edited by J. Henderson (E. and S. Livingstone).

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at the Children's Hospital, Melbourne, on April 11, 1932, DR. REGINALD WEBSTER, the President, in the chair.

Pink Disease.

DR. W. J. PENFOLD read a paper on some work on the ætiology of pink disease done at the Baker Institute of the Alfred Hospital in collaboration with Miss Butler and Dr. Ian Jeffreys Wood.

DR. J. W. GRIEVE showed a female infant, aged six months, who had been admitted to hospital with a history of irritability, anorexia, loss of weight and photophobia of two months' duration. A sudaminal rash had developed early and was well marked at the time of admission. The onset had been associated with fever and running at the nose. The baby had been born at full time, weighing 4·2 kilograms (nine and a quarter pounds), and had been breast-fed for three months; boiled cow's milk had then been given. From the age of six weeks he had received at least one teaspoonful of orange juice per day. The family history was irrelevant. At the time of admission to hospital the child presented irritability and photophobia, the nose was red and the hands and feet red and sodden, with a tendency to desquamation. The musculature was markedly atonic and the child adopted the characteristic attitude seen in pink disease. The weight was 5·6 kilograms (twelve pounds six ounces). A diet consisting of milk (three parts), water (one part), with added sugar (one drachm to every three fluid ounces) was given. Fifty-six cubic centimetres (two fluid ounces) of raw liver juice were given daily, together with orange juice, cod liver oil and the white of one raw egg. Progress was complicated by an ear infection, but the child improved a great deal, and at the time of the meeting was not irritable and weighed 6·2 kilograms (thirteen pounds six ounces). There were still some loss of tone and the characteristic appearance of hands and feet.

DR. G. M. TALLENT showed a patient suffering from erythrœdema, who was now better. The disease had developed when the child was three months old. The child was entirely breast fed, and at the age of six months, when its photophobia was most intense, the mother developed identical ocular symptoms.

DR. H. BOYD GRAHAM said he still thought pink disease was an avitaminosis. He had observed many patients who he thought were in the very early stage of erythrœdema, and they had become well when the diet had been adjusted and articles rich in the various vitamins had been added. More investigation along these lines was required.

DR. H. DOUGLAS STEPHENS thought that the disease was due to an infection and was not an avitaminosis. This was borne out by the fact that cases of the disease came in groups. In one year he and Dr. Hobill Cole had seen eighteen cases. The disease seemed to be more common in spring, and if it had not subsided by the end of the summer, many patients died of bronchopneumonia with the approach of colder weather. He had tried potato juice and yeast, but concluded that there was no specific as yet. In one case of his, of three months' duration, with extensive signs, antistreptococcal serum had caused a dramatic cure, but in many others it had been quite useless. In another he had tried "Radiostoleum", and this also had been followed by a dramatic cure, but again in many others it was of no avail.

DR. J. G. WHITAKER mentioned a patient of his who had been taken to the country and stayed in a house where there was a patient suffering from erythrœdema. Five weeks after returning to Melbourne typical erythrœdema had developed.

Dr. Penfold in reply said that in the German literature there had been a reference to acrodynia eight years before. He agreed with Dr. Graham that more thorough investigation of the rôle of vitamins was required, especially that of vitamin *B*; perhaps some new vitamin altogether was concerned. He leaned towards the infective theory himself and intended doing more work on the tonsils. Good results had been reported following tonsillectomy in many cases, and he hoped that the honorary medical staff of the Children's Hospital would let him have the tonsils removed in some of their typical cases, for further bacteriological research, or, failing this, as Dr. Webster had suggested, some culture material from tonsil puncture. He would like to see the disease made notifiable, perhaps through the influence of the British Medical Association, and then more thorough investigation of all aspects of the disease could be carried out.

Pontine Tumour.

DR. REGINALD WEBSTER showed a pontine tumour from a boy, aged ten years, who had been admitted to hospital on February 29, 1932, with the history that he had been well till December, 1931, when he had developed thickness in speech. He had consulted his doctor, who thought that his tonsils and adenoids were the cause. These had been removed in January, 1932. Secondary hæmorrhage had occurred on the fifth day, and convalescence had been very slow. Four weeks prior to admission there had been some weakness in both legs, and some difference of opinion had existed as to whether there was some facial weakness on the left side. Ten days before admission he had had a sudden seizure during the night, when he had awakened crying with headache and complaining of stiffness in the back of the neck. The temperature had risen, and the mother had thought that convulsions might be imminent. He had been very drowsy and vomited several times. In twenty-four hours he had recovered, but had been left with a definite left facial weakness and a staggering gait. The condition had then remained stationary except for the occurrence of mild attacks of stiffness in the back of the neck and a tendency to nasal regurgitation. The family and previous history were irrelevant. At the time of admission to hospital examination of his central nervous system revealed the following: (i) A left facial paresis of the peripheral type; (ii) weakness of the palatal and pharyngeal reflex, with drawling speech; (iii) left nerve

deafness; (iv) cerebellar signs, especially on the left side, such as nystagmus, hypotonia, pendulum knee jerk, awkwardness of movement, and a staggering gait; (v) pyramidal involvement on the right side, as shown by an extensor plantar reflex and poor response of the right superficial abdominal reflex.

The other systems were apparently normal. Examination of the fundi revealed no abnormality.

In hospital his condition became worse. He developed anæsthesia in the left cornea and then a palsy of the left external rectus muscle. He had frequent curious choking turns. Headaches were not a feature, and after admission he vomited only once. Slight papillœdema developed several weeks after admission. Dr. Webster remarked that the features of his illness had been the signs of involvement of the left cerebello-pontine angle. The differential diagnosis had lain between a true cerebello-pontine angle tumour of the *nervus acousticus* and a left-sided pontine tumour.

The patient died on April 13 while under general anæsthesia, before the skull could be opened. Dr. Leonard B. Cox reported that the tumour was an example of a glioma producing the so-called hypertrophy of the pons. It was an astrocytoma composed of protoplasmic astrocytes, hence its somewhat gelatinous rather than fibrous texture. It had apparently originated in the reticular substance on the floor of the fourth ventricle and must have involved most of the nuclei of the cranial nerves in this region. It had extended to the hilus of the dentate nucleus and must have involved both superior cerebellar peduncles. Anteriorly it was somewhat limited by the trapezoid bodies and the medial lemnisci, laterally by the middle cerebellar peduncles. Above it spread along the reticular substance and was still present to the left of the aqueduct in the upper part of the specimen.

Synovitis.

DR. J. G. WHITAKER presented a boy, aged eleven years, suffering from bilateral synovitis of the knees. There had been a swelling in each knee for the previous three years, which had increased with movement and largely disappeared during periods of rest. There had been conspicuous absence of pain throughout and no abnormal physical signs other than the marked effusion and the resultant slight ligamentous laxity. Muscular wasting and limitation were both absent. General examination of the boy revealed no other abnormality and there was complete absence of any stigmata of syphilis. The boy's and the parents' serum and the fluid from the joints did not react to the Wassermann test. Recent and old X ray appearances of the joints were all normal. Von Pirquet's reaction was strongly positive. The fluid was highly albuminous and contained many cells, both polymorphonuclear cells and lymphocytes. Injection of some of the fluid into a guinea-pig had not caused any apparent ill-effects after four weeks. A complete blood examination, including platelet count, coagulation and bleeding times, revealed no abnormality. The boy's heart was normal. Dr. Whitaker discussed the diagnosis and concluded with the observation that the examination of the fluid suggested an inflammatory basis, and the diagnosis so far must remain in doubt. A biopsy would probably be performed if the guinea-pig test yielded no information.

DR. H. C. COLVILLE said he had had a similar case. The patient was a girl, aged ten years. After two years this girl had been cured spontaneously, and was now completely normal. When syphilis had been excluded, he had suspected tuberculosis because there had obviously been active secretion and absorption. The patient had been treated by bandaging with Martin's elastic bandages to procure slight pressure. He suggested this line of treatment in Dr. Whitaker's case.

VICTORIAN MEDICAL BENEVOLENT ASSOCIATION.

THE ANNUAL MEETING OF THE VICTORIAN MEDICAL BENEVOLENT ASSOCIATION was held on March 9, 1932, SIR GEORGE CUSCADEN, the President, in the chair.

Office-Bearers.

The following officers for the year 1932 were elected:

President: Sir George Cuscaden.
Vice-Presidents: Dr. A. V. M. Anderson, Dr. R. H. Fetherston.
Honorary Secretaries: Dr. Edward L. Gault, Dr. Leonard Mitchell.
Honorary Treasurer: Sir George Cuscaden.
Members of Committee: Dr. G. T. Howard, Dr. W. G. Upjohn, Dr. W. S. Newton, Dr. Fay Maclure, and Dr. A. P. Derham.
Auditors: Messrs. R. A. Shackell and Son.

Annual Report and Balance Sheet.

The balance sheet presented by the Honorary Treasurer disclosed the following particulars.

The accumulated funds now stand at £7,716 11s. 11d. Of this amount the sum of £7,240 is invested in Commonwealth stock, Victorian Government stock, and Melbourne and Metropolitan Board of Works Inscribed stock. The balance is in the Commercial Bank at fixed deposit and current account and in the State Government Savings Bank.

The revenue for the year 1931 amounted to £468 5s. 3d. The sum of £114 5s. 9d. was distributed in relief and £353 19s. 6d. was added to accumulated funds.

In the report attention is drawn to the growing volume of claims, and members of the profession are asked to join the association, to which the annual subscription is five shillings, and thus associate themselves with a good work and assist in creating a large capital fund and a steadily growing revenue.

THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

THE following reports of the Honorary Secretary and the Honorary Treasurer of the Medical Benevolent Association of New South Wales for the year ended December 31, 1931, have been forwarded for publication.

Honorary Secretary's Report.

The closure of the Government Savings Bank of New South Wales greatly embarrassed the Council in its work of affording financial relief to necessitous members of the profession and their dependants. Fortunately a number of generous friends came to our assistance; and before the end of the year our position was again secure. We especially wish to thank Mrs. Murray Oram and Mrs. R. L. Faithfull for liberal help in answer to requests for assistance.

The Council desires to bring before the members an addition to the Rules allowing of Honorary Members being elected.

The Council suggests that those who are already members of the Association might call the attention of their friends to the advantage of membership. We should have a great many more members, and no doubt will have when the profession at large becomes better acquainted with the beneficent nature of our activities.

J. M. GILL,
Honorary Secretary.

Honorary Treasurer's Report.

As will be seen from the balance sheet, our assistance to beneficiaries was somewhat curtailed this year, and necessarily so, owing to the closure of the Government Savings Bank of New South Wales, in which were all our liquid assets.

However, thanks to the energy of our Honorary Secretary and the liberal response of many members of the profession to his appeal for help, we were enabled to supply, at any rate, the necessaries of life. Later also, owing to the generosity of many members in reply to our Christmas appeal, we were able to retrieve the position somewhat by substantial and extremely welcome Christmas gifts. We beg to thank all those members who so kindly

THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

Balance Sheet as at December 31, 1931.

LIABILITIES.	£	s.	d.	ASSETS.	£	s.	d.
Capital Account as at December 31, 1930 ..	1,925	13	6	Investments—			
Add Surplus for Year Ended December, 1931, as per Income and Expenditure Account ..	162	10	8	Australian Commonwealth Consolidated Inscribed Stock, 4%	1,400	0	0
				Cash Accounts—			
				Commonwealth Savings Bank of Australia—			
				Current Account, General Account £30 10 3			
				Christmas Fund 46 3 9			
					76	14	0
				Commonwealth Savings Bank of Australia Deposit Accounts	30	3	7
				Government Savings Bank of New South Wales, Old Business—	32	6	7
				Deposit Account	267	0	0
				Current Account	282	0	0
	£2,088	4	2		£2,088	4	2

Income and Expenditure Account for Year ending December, 1931.

EXPENDITURE.	£	s.	d.	INCOME.	£	s.	d.
To Assistance to Beneficiaries	375	12	0	By Donations	55	10	0
„ Christmas Gifts to Beneficiaries	45	0	0	„ Annual Subscriptions (234)	245	14	0
„ Printing and Stationery	17	9	4	„ Life Subscriptions (8)	84	0	0
„ Stamps	19	5	11	„ Exchange added	1	4	0
„ Exchange	1	15	6	„ Interest on Investments	116	12	5
„ Glass and Brass Plates for Door	1	6	6	„ Christmas Fund Appeal	116	17	6
„ Typing	1	10	0	„ Balance, Honorary Secretary's Account, brought forward	78	3	0
„ Honorary Secretary's Unexpended Balance carried forward	73	11	0				
„ Surplus for year added to Capital Account	162	10	8				
	£698	0	11		£698	0	11

M. O'GORMAN HUGHES,
RAYMOND GREEN,
Honorary Auditors.

E. S. LITTLEJOHN,
Honorary Treasurer.

January 1, 1932.

came to our rescue at a time of great difficulty. We have also to express our gratitude to the Illawarra Suburbs Medical Association and the Northern District Medical Association for their welcome donations.

It is very gratifying to find that, notwithstanding the adverse conditions, we have finished the year with a surplus of £162 10s. 8d., of which £46 3s. 9d. belongs to the Christmas Gift Fund.

Our great need is still for many more annual members; in a profession numbering over 1,700 members of the British Medical Association we have only 234 annual subscribers.

E. S. LITTLEJOHN,
Honorary Treasurer.

Correspondence.

"AVERTIN."

SIR: Your leader on "Avertin" in THE MEDICAL JOURNAL OF AUSTRALIA of May 21, and the implied unfavourable criticism of its use for anæsthesia, compels me to reply, even though I am very averse to journalistic argument.

I saw "Avertin" used in Germany in 1927, at the time when the anæsthetists of the Rudolph Virchow Hospital, Berlin, had used it in over five hundred cases. These anæsthetists were very satisfied with it, and my observations of its use led me to become very interested. The manufacturers, on being satisfied of its safety and value by virtue of trial by their own appointed surgeons, made it available to the profession. They sent a trial supply to me in Australia, as they had promised, and this I received in August, 1929. I did not attempt to use it until I had a surgical case for operation in which a trial use was justified. This was a case of carcinoma of the buccal end of the œsophagus, with practically total obstruction and with gross pressure congestion of the pharyngeal and laryngeal mucosa, for which inhalation anæsthesia was contraindicated. This was in December, 1929. After free and frank discussion the patient agreed to the use of "Avertin" for anæsthesia. This case was such a success that I began to use it for further cases, always with the patient's knowledge and consent. Since then, over a period of two and a half years, I have practically used no other general anæsthetic, and I have not yet found "Avertin" cause me anxiety or dissatisfaction.

I have been associated with its use, as the operator chiefly, and also as the anæsthetist, in over three hundred cases included in my public hospital and in my private practice. The private practice has been spread over at least eleven private hospitals in Melbourne, and the public hospital work has been done at the Queen Victoria Hospital. My opinion, that the worse the "anæsthetic risk" of the patient's condition, the more the use of "Avertin"

is called for, has been forced upon me by experience, and I believe that I see the pharmacological and physiological reason for this benefit.

A few examples out of many:

1. The case of adeno-carcinoma of kidney (done in 1930) reported by me in your journal last year (June 20, 1930). This patient was shown at the British Medical Association clinical meeting at the Queen Victoria Hospital this year.

2. A case of moribund *purpura hæmorrhagica* with enlarged spleen and naso-pharyngeal bleeding. I was begged to do this case, which had already had two transfusions, was still bleeding profusely from the nose, was moribund and had a cadaveric odour. After long deliberation, when the patient was brought to the operating theatre, I decided to shoulder the responsibility to give the girl a chance under "Avertin" anæsthesia. Inhalation anæsthesia was out of the question owing to the naso-pharyngeal bleeding, and both nostrils were plugged on the operating table. I performed the operation of splenectomy and also a transfusion. The anæsthesia was perfect and free of any anxiety. The patient made a marvellous recovery, and was shown by Dr. K. Mackay at the Queen Victoria Hospital clinical meeting.

3. A case of cholelithiasis, acute cholecystitis with *icterus gravis* and a fibrillating heart. I had attended this patient off and on over a period of four years for attacks of acute pulmonary œdema of cardiac origin. She had not had an attack for twelve months before this attack of cholecystitis, in which she was first seen by her lodge doctor and two *locum tenentes* and later by another surgeon. She was advised that an operation was necessary to save her life, but that as her condition was so precarious, no surgeon would attempt it. In this desperate condition I was appealed to by the patient and by her people, and after all risks had been fully placed before and accepted by them, I operated under "Avertin" anæsthesia. Under this anæsthesia, which was perfect, the pulse force and volume improved and the rate slowed nicely. The operation, cholecystostomy, after removal of gall-stones and freeing of the common bile duct, which was twisted spirally by inflammation and adhesions, was tedious, but the patient did not "turn a hair". The liver showed evidence of cholangitis, and the gall-bladder was very inflamed and friable. Convalescence was uninterrupted and the patient was discharged well in six weeks. As the other doctors present said, this case proved the value and benefits of "Avertin". This operation and the following one were done at the Jessie McPherson Community Hospital, Melbourne.

4. A case of thyreotoxicosis with a basal metabolic rate of 51 after some months of medical treatment. This patient left the operating table with a pulse of 120, had a hyperthyreoidic reaction of medium degree between twenty and thirty hours after operation; then made a very good recovery and left hospital two and a half weeks after operation, with a pulse of 76. The experienced physician who referred this case to me, and who supervised the "Avertin" anæsthesia, is of the opinion that "Avertin" had given the patient her best chance. At practically all of these 300 cases other independent doctors have been present as supervisors or assistants, and in not one case has any death occurred under or been attributable to the "Avertin". Of course, as with any anæsthetic, the skill and experience of the anæsthetist is a big factor, and lack of these may have contributed to the trouble you report, for which the anæsthetic has to carry the adverse criticism.

I commenced practice as resident medical officer at the Melbourne Hospital and have at various times administered and operated under: (i) rectal oil and ether, (ii) intratracheal ether, (iii) gas and oxygen, (iv) ethylene, (v) ether, (vi) chloroform and chloroform-ether mixture; and in my opinion, based on experience and observation: (*a*) "Avertin" is as far ahead of these as a motor car is ahead of a bicycle, from the points of view of the surgeon, anæsthetist, patient and nursing sisters; (*b*) no patient need be refused its benefits.

I advance this contribution based on personal experience of cases, and I think it only fair that it may be demanded of any who wish to adversely criticize the use of "Avertin" for anæsthesia, that they state: (i) the complete extent of their practical experience with it; (ii) from whom was the tuition of its use received.

There is another aspect of safety that is often lost sight of, and that is the ability of the anæsthesia to facilitate or impair the patient's actual physical and visceral conditions under which the surgeon does the actual work.

In extraordinarily and even in ordinarily difficult surgical crises, one or other condition of venous congestion, restlessness, tendency to vomit, heaving of the abdominal contents and insufficient relaxation, often mars the effectiveness of the operative technique and resulting benefit to the patient. I do not mean to suggest that these conditions pertain in other anæsthetics all the time, but they do occur, as surgeons do know, and have a tendency to do so at critical times. Under "Avertin" anæsthesia operative technique gets its fullest scope; for there is no congestion, no bowel movement, no distension, no abdominal heaving, no vomiting, no restlessness; the abdominal contents just lie quiet and passive, and the musculature is in a state of full relaxation. No post-operative retching or distension ensues to affect the union of the abdominal wound.

Another medico-psychological benefit is that instead of the practice of surgery sending the patient home loaded with fear of any future surgical procedure, it can send the patient home mentally happy in that almost invariably I am told: "Well, Doctor, if I can have 'Avertin', I would not be frightened or anxious if I had to have a dozen operations."

On the foregoing I claim that my opinion and choice of "Avertin" for anæsthesia is justified.

The details of the use of "Avertin" as published by me in THE MEDICAL JOURNAL OF AUSTRALIA, April 19, 1930, and June 20, 1931, stand as true now as they were when reported.

ELLEN M. BALAAM, M.B., B.S.,
Honorary Surgeon to In-Patients, Queen Victoria Hospital, Melbourne.

May 25, 1932.

THE ROBERT H. TODD PRIZE IN MEDICAL JURISPRUDENCE.

ADDITIONAL subscriptions towards the Robert H. Todd Prize in Medical Jurisprudence have been received as follows:

£5 5s.: Dr. S. A. Smith.

£1 1s.: Dr. H. G. Chapman, Dr. F. Brown Craig, Dr. B. T. Edye, Dr. Cotter Harvey, Dr. H. G. Holmes, Dr. H. E. Lee, Dr. H. K. Porter.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered pursuant to the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

Evans, Robert, M.B., B.Ch., B.A.O., N.U., 1924 (Ireland), Toowoomba.

Murray, Robert Malcolm, M.B., B.S., 1928 (Univ. Sydney), Townsville.

Cameron, Donald Alastair, M.B., B.S., 1927 (Univ. Sydney), Ipswich.

Murray, Ronald Elliott, M.B., 1927 (Univ. Sydney), Palm Island.

Restoration to the Register:

Casey, Edward William, M.B., B.S., 1925 (Univ. Melbourne), Brisbane.

Additional diplomas:

Elwell, Laurence Bedford, M.R.C.P., 1931 (London), M.R.C.P., 1932 (Edinburgh).

Obituary.

WILLIAM ROBERT MATHEWS.

We regret to announce the death of Dr. William Robert Mathews, which occurred on May 23, 1932.

Books Received.

PHYSICIANS' MANUAL OF BIRTH CONTROL, by A. F. Konikow, M.D.; 1931. London: Baillière, Tindall and Cox. Royal 8vo., pp. 258, with 21 illustrations. Price: 12s. 6d. net.

SELECTED WRITINGS OF JOHN HUGHLINGS JACKSON; Volume II: Evolution and Dissolution of the Nervous System, Speech, Various Papers, Addresses and Lectures, edited by James Taylor, M.D., F.R.C.P., with the advice and assistance of G. Holmes, M.D., F.R.C.P., and F. M. R. Walshe, M.D., F.R.C.P.; 1932. London: Hodder and Stoughton Limited. Royal 8vo., pp. 518. Price: 25s. net.

THE HEART AND SPLEEN IN HEALTH AND DISEASE, by G. A. Stephens, M.D., B.S., B.Sc.; 1932. London: H. K. Lewis and Company, Limited. Demy 8vo., pp. 152, with six illustrations. Price: 7s. 6d. net.

MINOR MONOGRAPH SERIES: LIGHTNING, LIGHTNING-STROKE AND ITS TREATMENT, by H. A. Spencer, M.R.C.S., L.R.C.P.; 1932. London: Baillière, Tindall and Cox. Crown 8vo., pp. 100. Price: 5s. net.

Diary for the Month.

JUNE 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 10.—Queensland Branch, B.M.A.: Council.
JUNE 14.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 15.—Western Australian Branch, B.M.A.: Branch.
JUNE 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 22.—Victorian Branch, B.M.A.: Council.
JUNE 24.—Queensland Branch, B.M.A.: Council.
JUNE 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 30.—South Australian Branch, B.M.A.: Branch.
JUNE 30.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. C. N. Raphael (B.M.A.) has been appointed Public Vaccinator for Fitzroy, Victoria.

* * *

Dr. R. A. Maxwell (B.M.A.) has been appointed Government Medical Officer at Aramac, Queensland.

* * *

Dr. A. E. Burke-Gaffney has been appointed Government Medical Officer at Cooktown, Queensland, and a Health Officer under *The Health Acts, 1900 to 1931.*

* * *

Dr. C. Gurner (B.M.A.) has been appointed Honorary Assistant Deep X Ray Therapist at the Adelaide Hospital, South Australia.

* * *

The undermentioned appointments have been made at the Royal Alexandra Hospital for Children, Camperdown, New South Wales: Honorary Assistant Physician, Dr. R. J. Taylor (B.M.A.); Honorary Relieving Assistant Physician, Dr. L. Dods (B.M.A.); Honorary Dermatologist, Dr. G. Norrie (B.M.A.).

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

LAUNCESTON PUBLIC HOSPITAL, TASMANIA: Resident Medical Officer (male).
THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

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